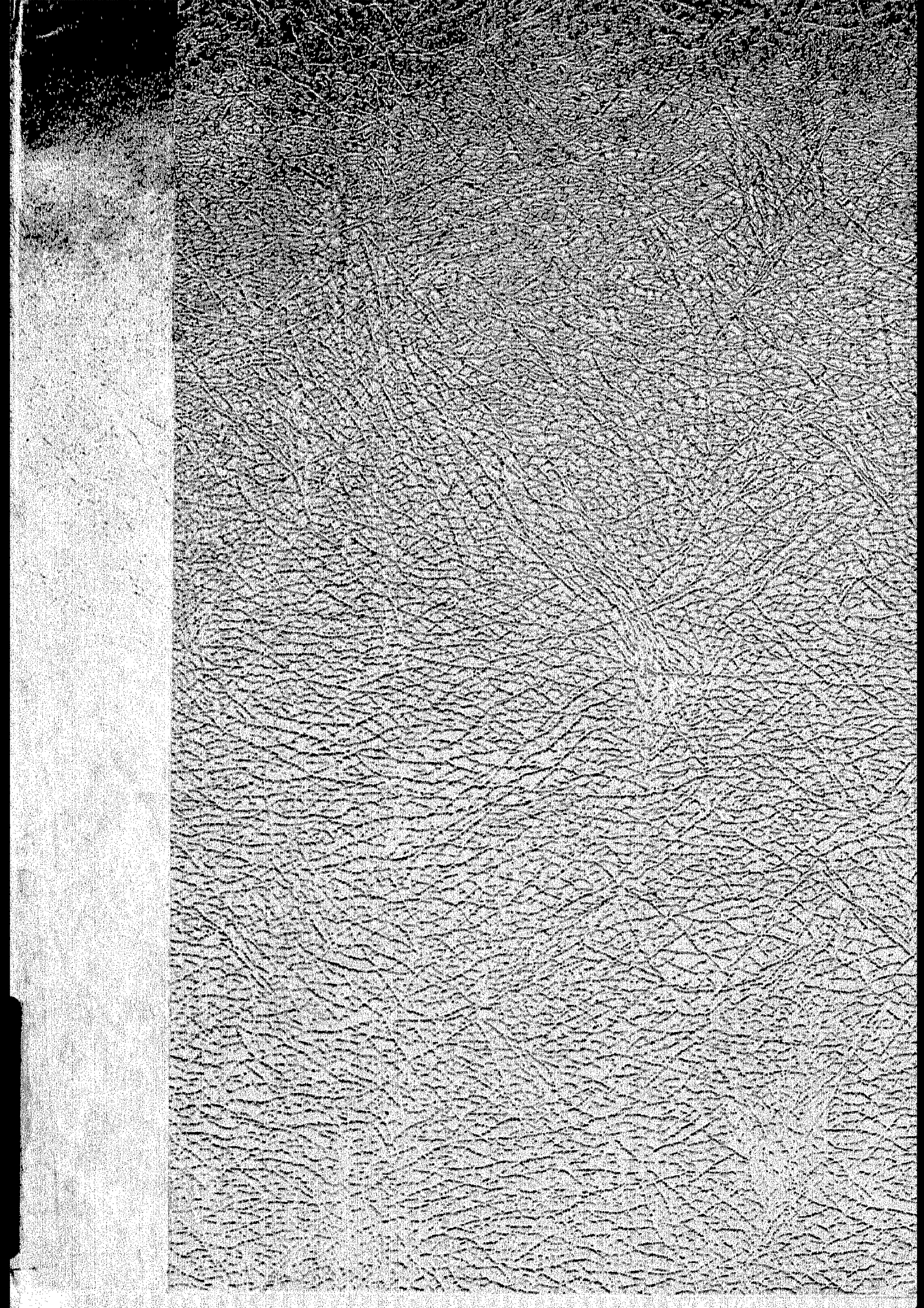


INTRA-OPERATIVE ARRHYTHMIAS AETIOLOGY AND MANAGEMENT

Essay

Submitted for partial fulfillment of
Master Degree in Anaesthesia

By

Osama Mohamed Ghonemy
M.B.,B.Ch.

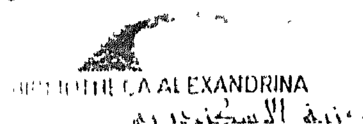
Supervisors

Prof. Dr. Yahia Abdel Raheim Hememy
Prof. of Anaesthesia and ICU
Faculty of Medicine
Ain Shams University

Prof. Dr. Magdy Mohamed Houssein Nafei
Assist. Prof. of Anaesthesia and ICU
Faculty of Medicine
Ain Shams University

Dr. Hesham Mohamed Mahmoud Tahoun
Lecturer of Anaesthesia and ICU
Faculty of Medicine
Ain Shams University

Faculty of Medicine
Ain Shams University
2002



بسم الله الرحمن الرحيم

﴿قَالُوا سُبْحَانَكَ لاَ عِلْمَ لَنَا إلاَّ مَا عَلَّمْتَنَا إِنَّكَ أنتَ الْعَلِيمُ الْحَكِيمُ﴾

صدق الله العظيم

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Introduction

INTRODUCTION

Cardiac arrhythmias may be defined as irregular or abnormal Rhythms, by convention, they also include bradycardias or tachycardias outside the physiological range. (i.e. 55-90 beats/min in adults). Cardiac arrhythmias occur frequently during anaesthesia and surgery, most of them are of short duration, compromise the circulation minimally and do not require pharmacological interventions. However, in poor risk patients, cardiac arrhythmias may seriously compromise cardiac output and cause life threatening complications that require active treatment **(Fleisher, 2000)**

Cardiac arrhythmias occur most commonly during endo tracheal intubation, extubation and more frequently in patients with preexisting cardiac disease under going non cardiac and cardiac surgery. The broad principles of the anaesthetic management of such cases include: proper assessment proper investigation of the patient and adequate monitoring of the case not only to detect early or serious arrhythmias but also to check out the probable causes of the ectopies (**Thomas and Kramer 1993**).

Chapter I

Chapter I

ANATOMY OF CONDUCTIVE SYSTEM

The heart normally beats in orderly sequence: contraction of the atria **(atrial systole)** is followed by contraction of the ventricles **(ventricular systole)**, and during diastole all four chambers are relaxed. Heart beat originates in a specialized cardiac conduction system and spreads via this system to all parts of the myocardium. The structures that make up the conduction system are the sinoatrial node (SA node), the internodal pathways, the atrioventricular node (AV node), the bundle of His with its branches, and the Purkinje system. However, SA node normally discharges most rapidly and depolarization spreads from it to the other regions. The SA node is therefore the normal cardiac pacemaker, its rate of discharge determining the rate at which the heart beats. Impulses generated in the SA node pass through the atrial pathways to the AV node, through this node to the bundle of His, and through the branches of the bundle of His via the Purkinje system to the ventricular muscle **(Nayler et al., 1984)**.

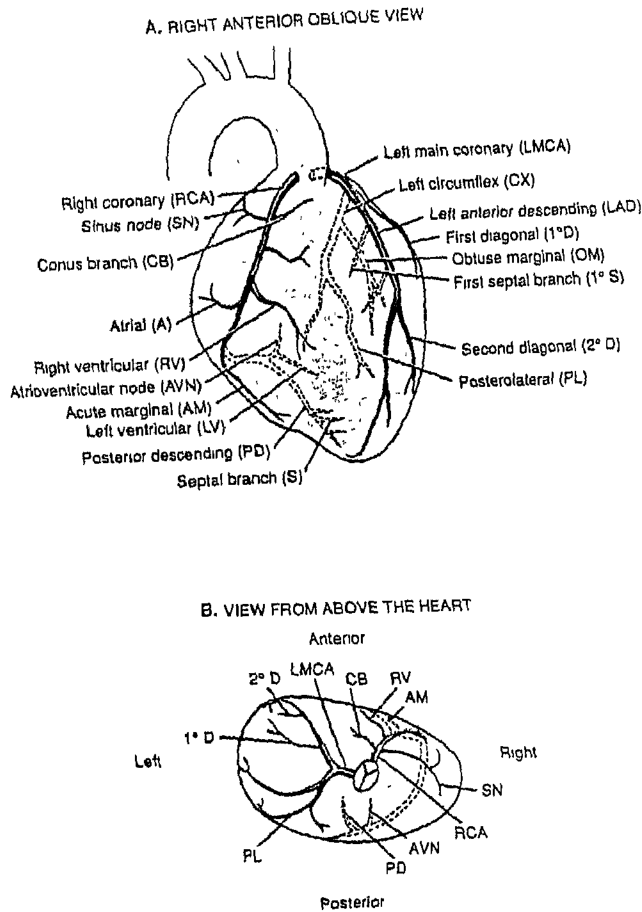


Fig. (I): Anatomy of the coronary arteries. A: Right anterior obliqu view. B. View from above.

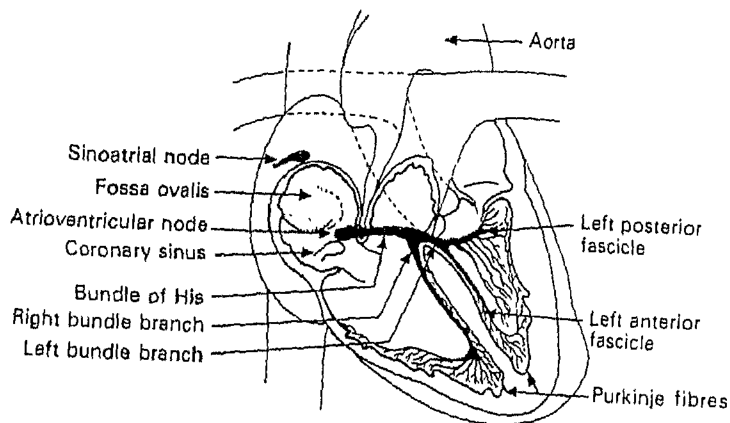


Fig. (II): The cardiac conducting system.

Anatomic Considerations

In the human heart, the SA node is located at the junction of the superior vena cava with the right atrium. The AV node is located in the right posterior portion of the interatrial septum. There are bundles of atrial fibers that contain Purkinje type fibers and connect the SA node to the AV node: the anterior internodal tract of Bachman, the middle nodal tract of Wenckebach, and the posterior in-ernodal tract of Thorel. However, there is debate about the role of these bundles versus the regular atrial myocytes in conduction from the SA node to the AV node **(Katz, 1975)**.

The AV node is normally the only conducting pathway between the atria and ventricles. It is continuous with the bundle of His, which gives off a left bundle branch at the top of the interventricular septum and continues as the right bundle branch. The left bundle branch divides into an anterior fascicle and a posterior fascicle. The branches and fascicles run subendocardially down either side of the septum and come into contact with the purkinje system, whose fibers spread to all parts of the ventricular myocardium **(Nayler et al., 1984)**.

The conduction system is composed of the most part of modified cardiac muscle that has fewer striations and indistinct boundaries. The SA node and, to a lesser extent, the AV node, also contain small round cells with few

organelles which are connected by gap junctions. These are probably the actual pacemaker cells, and therefore they are called **P** cells. The atrial muscle fibers are separated from those of the ventricles by a fibrous tissue ring and normally the only conducting tissue between the atria and ventricles is the bundle of His **(Nayler et al., 1984).**

Blood Supply

- The blood supply to the SA node is from the right coronary artery (R.C.A) in 55 percent of patients and left circumflex coronary artery (C.C.A) in 45 percent of patients **(James 1961).**
- The AV node blood supply comes from the RCA in 85 to 90 percent of patients and from CCA in remainder.
- The left bundle is supplied by both the RCA and left anterior descending (LAD) coronary artery and so is relatively protected from ischemia **(Frink and Jaones, 1973).**
- The right bundle branch (RBB) and the anterior division of the left bundle branch are supplied by LAD. Where as the posterior division of the L.B.B may be supplied by either the RCA or the LAD. **(Levine 1993)**

Innervation of conductive system:

- The sympathetic innervation of the heart arises from the sympathetic nerves originating in the thoracic segment T^1 to T^4 .

Sympathetic effects are mediated by norepinephrine release at the postganglionic nerve terminals and by circulating catecholamines.

Catecholamines increase pacemaker discharge rate, shorten AV node conduction time and decrease resistance to subsequent excitation (refractoriness) but do not affect normal His bundle conduction **(Witt, et al., 1975)**.

The SA node develops from structures on the right side of the embryo and the AV node from structures on the left side. This is why in the adult right vagus is distributed mainly to the SA node and the left vagus mainly to the AV node.

Similarly, the sympathetic innervation on the right side is distributed primarily to the SA node and the sympathetic innervation on the left side primarily to the AV node. On each side, most sympathetic fibers come from the stellate ganglion. Noradrenergic fibers are epicardial, whereas the vagal fibers are endocardial. However, connections exist for reciprocal inhibitory effects of the sympathetic and parasympathetic innervation of the heart on each other **(Witt, et al., 1975)**.

Chapter II

Chapter II

PHYSIOLOGY OF CONDUCTIVE SYSTEM

Pacemaker Potentials

Rhythmically discharging cells have a membrane potential that after each impulse declines to the firing level Thus, this prepotential or pacemaker potential (Figure 1) triggers the next impulse. At the peak of each impulse, potassium current (I_k) begins and brings about repolarization. I_k then declines, and as K^+ efflux decreases, the membrane begins to depolarize, forming the first part of the prepotential. Ca^{2+} channels then open. Calcium channels are of two types in the heart, the **T** (for transient) channels and the **L** (for long-lasting) channels. The calcium current (I_{ca}) due to opening of T channels completes the prepotential, and I_{ca} due to opening of **L** channels produces the impulse **(Ross, 1983)**.

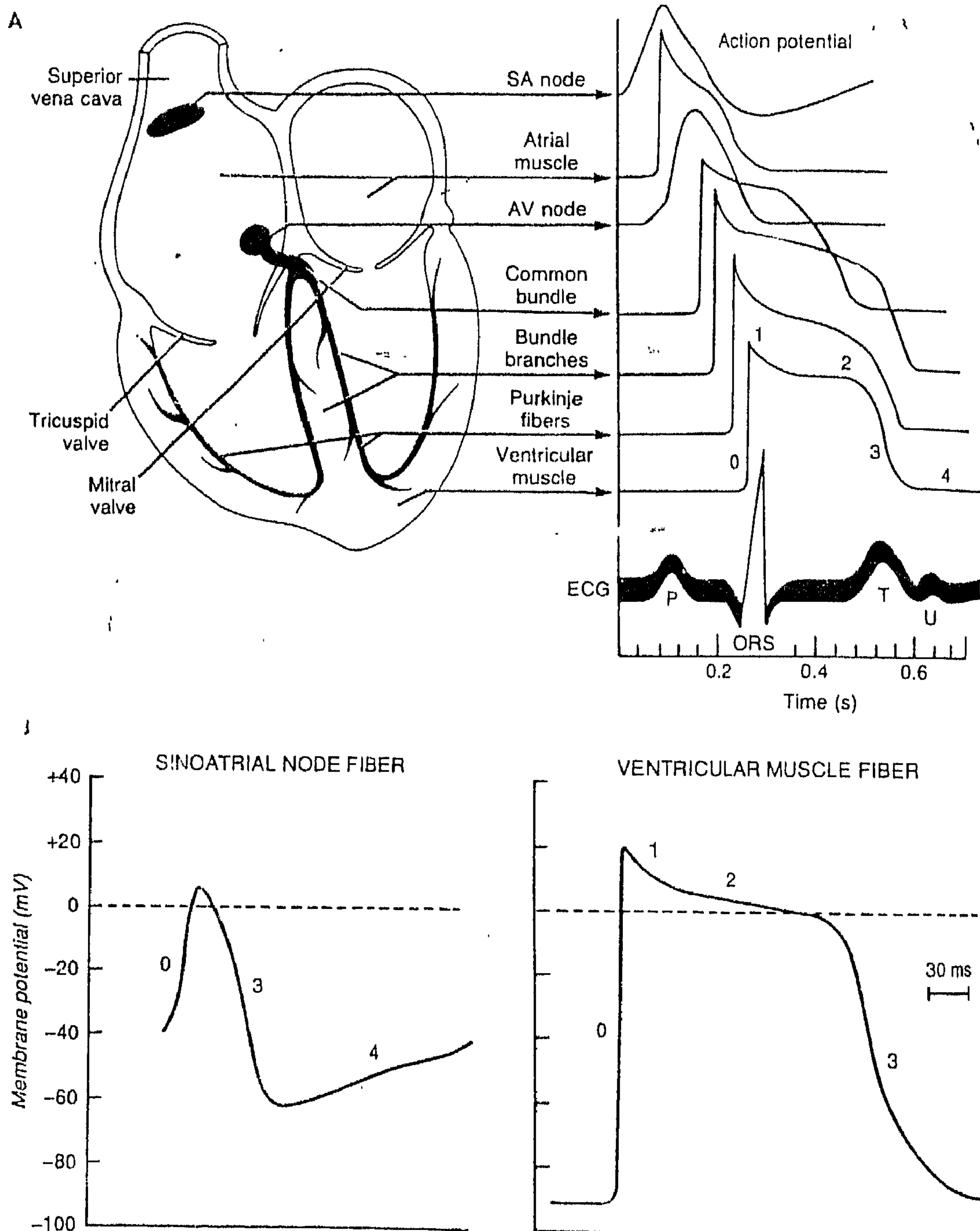


Fig. (1): Cardiac action potentials. A: Note the characteristic action potentials of different parts of the heart. B: Pacemaker cells in the SA node lack the same distinct phases as atrial and ventricular muscle cells and display prominent spontaneous diastolic depolarization *(Ganong, 1993)*..

The action potentials in the SA and AV nodes are largely due to Ca^{2+}, with little contribution of Na^+ influx. Consequently there is no sharp, rapid depolarizing spike before the plateau, as there is in other parts of the conduction system, the artial and the ventricular fibers. In addition, prepotentials are normally prominent only in the SA and AV nodes **(Rusy, 1987)**.

However, there are "latent pacemakers" in other portions of the conductive system that can take over when the SA and AV nodes are depressed or conduction from them is blocked. Atrial and ventricular muscle fibers do not have prepotentials discharge spontaneously only when injured or in abnormal condition **(Ross, 1987)**.

When the cholinergic vagal fibers to nodal tissue are stimulated, the membrane becomes hyperpolarized and the slope of the prepotentials is decreased (Figure 2) because the acetylcholine released at the nerve endings Increases the K^+ conductance of nodal tissue. This action is mediated by M_2 muscarinic receptors, which, act, via G protein that decreases, cAMP in the cells, slows the opening of the Ca^{2+} channels and decrease the firing rate. Strong vagal stimulation may abolish spontaneous discharge for some time **(Rusy, 1987)**.

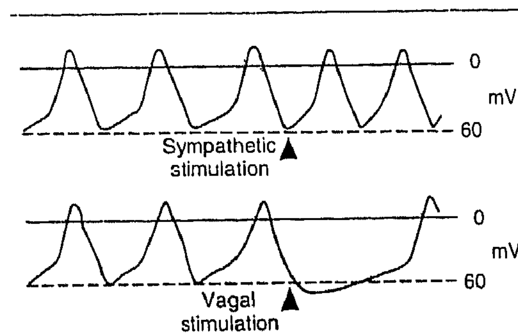


Fig. (2): Effect of sympathetic (noradrenergic) and vagal (cholinergic) stimulation on the membrane potential of the SA node.

Conversely, stimulation of the sympathetic cardiac nerves makes the membrane potential fall more rapidly, and the rate of spontaneous discharge increases. Norepinephrine secreted by the sympathetic endings binds to β-receptors, and the resulting increase in intracellular cAMP facilitates the opening of the channels, increasing I_{ca} and the rapidity of the depolarization phase of the impulse. Because of the "sidedness" of the cardiac innervation, stimulation of the right vagus slows the heart by inhibiting the SA node, whereas stimulation of the left vagus mainly slows AV conduction, stimulation of the right stellate ganglion accelerates the heart, whereas stimulation of the left stellate ganglion shortens the AV nodal conduction time and refractoriness **(Feld et al., 1992)**.

The rate of discharge of SA node and other nodal tissue is influenced. by temperature and drugs. The discharge frequency is increased when the temperature rises, and this may contribute to the tachycardia associated

with fever. Digitalis depresses nodal tissue and on the other hand exerts a stimulatory effect, particularly on the AV node **(Hurst et al., 1990)**.

Spread of Cardiac Excitation

Depolarization initiated in the SA node spreads radially through the atria, then converges on the AV node. Atrial depolarization is complete in about 0.1 s. Because conduction in the AV node is slow there is a delay of about 0.1 sec. (AV nodal delay) before excitation spreads to the ventricles. This delay is shortened by stimulation of the sympathetic nerves to the heart and lengthened by stimulation of the vagi. From the top of the septum the wave of depolarization spreads in the rapidly conducing Purkinje fibers to all parts of the ventricles in 0.08-0.1 sec **(Ohkawa et al., 1991)**.

In humans, depolarization of the ventricular muscle starts at the left side of the interventricular septum and moves first to the right across the midportion of the septum. The wave of: depolarization then spreads down the septum to the apex of the heart. The last parts of the heart to be depolarized are the posterobasal portion of the left ventricle, the pulmonary conus and the uppermost portion of the septum **(Bigger, 1984)**.

Cardiac muscle Resting Membrane and Action Potentials

The resting membrane potential of individual mammalian cardiac muscle cells is about -90 mV (interior negative to exterior). Stimulation produces a propagated action potential that is responsible for initiating contraction. Depolarization proceeds rapidly and an overshoot is present, as in skeletal muscle and nerve but this is followed by a plateau before the membrane potential returns to the baseline **(Smeets et al., 1986)**.

In mammalian hearts depolarization lasts about 2 ms, but the plateau phase and repolarization last 200 ms or more. Repolarization is therefore not complete until the contraction is half over. Resting membrane potential of cardiac muscle is affected by external K^+ concentration, whereas changes in the external Na+ concentration affect the magnitude of the action potential The initial rapid depolarization and the overshoot (phase 0) are due to opening of voltage-gated Na^+ channels similar to that occurring in nerve and skeletal muscle. The initial rapid repolarization (phase 1) is due to closure of Na^+ channels. The subsequent prolonged plateau (phase 2) is due to a slower but prolonged opening of voltage-gated Ca^{2+} channels. Final repolarization (phase 3) to the resting membrane potential (phase 4) is due to closure of the Ca^{2+} channels and K^+ efflux through various types of K^+ channels **(Suga, 1990)**.

Voltage-gated Na^+ channel in cardiac muscle has two gates: an outer gate that opens at start of depolarization, at a membrane potential of -70 to-80 mV; and an inner gate that closes and preclude further influx until the action potential is over (Na^+ channel inactivation). In cardiac muscle slow Ca^{2+} channel is activated at a membrane potential of -30 to -40 mV. In addition there are three types of K^+ channels that produce repolarization. The first produces a transient early out ward current (I_{To}) that produces an early incomplete repolarization. The second is inwardly rectifying, ie at plateau potentials it allows K^+ influx but resists K^+ efflux and only at lower membrane potentials does it permit K^+ efflux The current it produces is called I_{kr}.

The third type is a slowly activating (delayed rectifying) type that produces a current called I_{ks}. The sum of I_{kr} and I_{ks} is a small net outward current that increases with time and produces repolarization **(Entman et al., 1990)**.

Table (1): cardiac ion channels (Smeets et al., 1986)

Voltage-gated channels[2]

- Na^+
- T Ca^{2+}
- L Ca^{2+}

K^+ channels

- Inward rectifying
- Delayed rectifying
- Transient outward
- Ligand-gated K^+ channels
  - Ca^{2+}-activated
  - Na^+ -activated
  - ATP-sensitive
  - Acetylcholine-activated
  - Arachidonic acid-activated

The Electrocardiogram

Because the body fluids are good conductors, the action potentials of myocardial fibers can be recorded extracellularly. These recorded potentials during the cardiac cycle are the electrocardiogram (ECG). Most electrocardiograph machines record these potentials on a moving stirp of paper. The ECG may be recorded by using an active or exploring electrode connected to an indifferent electrode at zero potential (unipolar recording), or by using two active electrodes (bipolar recording).

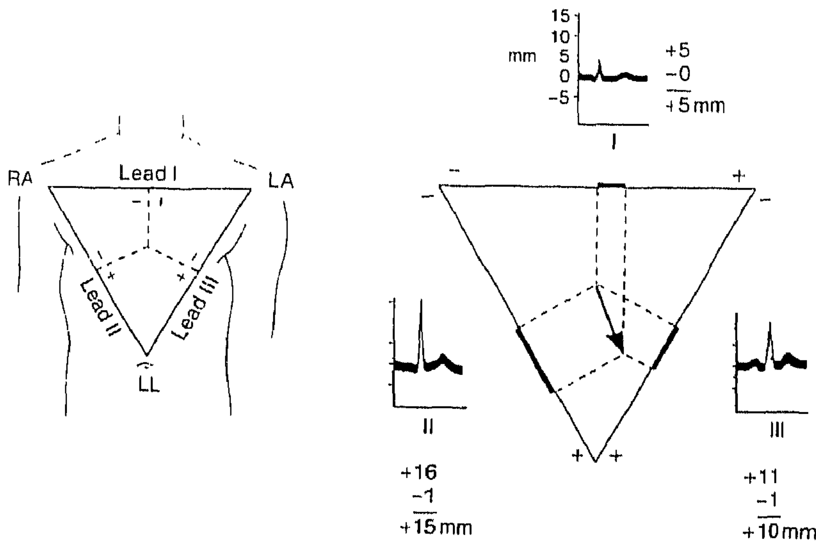


Fig. (3): Cardiac vector. **Left:** Einthoven's triangle. Perpendiculars dropped from the midpoints of the sides of the equilateral triangle intersect at the center of electrical activity. RA, right arm; LA, left arm; LL, left leg, **Center:** Calculation of mean QRS vector. In each lead, distances equal to the height of the R wave minus the height of the largest negative deflection in the QRS complex are measured off from the midpoint of the side of the triangle representing that lead. An arrow drawn from the center of electrical activity to the point of intersection of perpendiculars extended from the distances measured off on the sides represents the magnitude and direction of the mean QRS vector. **Right:** Reference axes for determining the direction of the vector *(Ganon, 1997).*

In a volume conductor, the sum of the potentials at the points of an equilateral triangle with a current source in the center is zero at all times. A triangle with the heart at its center. **Einthoven's triangle** can be approximated by placing electrodes on both arms and on the left leg. These are the three **standard limb leads** used in electrocardiography. If these electrodes are connected to a common terminal, an indifferent electrode that stays near zero potential is obtained. Depolarization moving toward an active electrode in a volume conductor produces a positive deflection, whereas depolarization moving in the opposite direction produces a negative deflection **(Weidmann, 1974).**

In the ECG the **P** wave is produced by atrial depolarization the QRS complex by ventricular depolarization, the ST segment and **T** wave by ventricular repolarization. The manifestations of atrial repolarization are not normally seen because they are obscured by the QRS complex The U wave is an inconstant finding, believed to be due to slow repolarization of the papillary muscles The intervals between the various waves of the ECG and the events in the heart that occur during these intervals are shown in table (2) **(Brodsky et al., 1997).**

Bipolar leads

Bipolar leads were used before unipolar leads were developed. The **standard limb leads**: leads **I II,** and **III,** each record the differences in potential between two limbs. Since current flows only in the body fluids, the recordings obtained are those that would be obtained if the electrodes were at the points of attachment of the limbs, no matter where on the limbs the electrodes. In lead I, the electrodes are connected so that an upward deflection is inscribed when the left arm becomes positive relative to the right (left arm positive). In lead II, the electrodes are on the right arm and left leg, with the leg positive; and in lead III, the electrodes are on the left arm and left leg, with the leg positive **(Brodsky et al., 1997).**

Table (2): ECG intervals

Interval	Normal duration(s)		Events in the during interval
	Average	Range	
PR Interval[1]	0.18[2]	0.12 - 0.20	Atrial depolarization and conduction through AV node
ORS duration	0.08	To 0.10	Ventricular depolarization and atrial repolarization
QT interval	0.40	To 0.43	Ventricular depolarization plus ventricular repolarization
ST interval (QT minus QRS)	0.42	....	Ventricular repolarization

1. Measured from the beginning of the P wave to the beginning of the QRS complex .
2. Shortens as heart rate increases from average of 0.18 at a rate of of 70 beats/min to 0.14 at a rate of 130 beats/min are placed *(Hurst et al., 1990)* .

Unipolar (V) Leads

An additional nine unipolar leads, ie leads that record the potential difference between an exploring electrode and an indifferent electrode. There are six unipolar chest leads (precordial leads) designated V_1-V_6 (Figure 4) and three unipolar limb leads: VR (right arm). VL (left arm), and VF (left foot). Augmented limb leads, designated by the letter a (aVR, aVL, aVF), are generally used. The augmented limb leads are recordings between one limb and the other two limbs. This increases the size of the potentials by 50 % without any change in configuration from the nonaugmented record. Unipolar leads can also be placed at

the tips of catheters and inserted into the esophagus or heart **(Meda et al., 1983).**

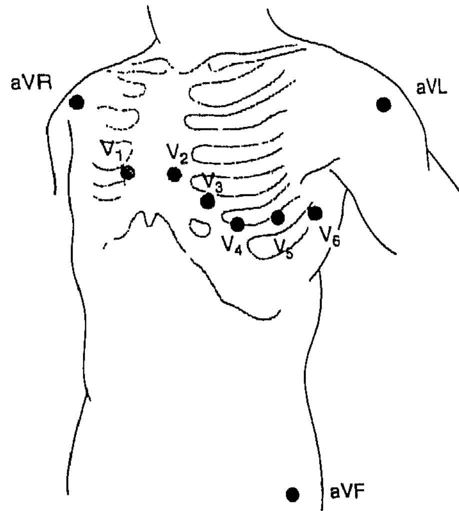


Fig. (4): Unipolar electrocardiographic leads *(Hurst et al., 1990)* .

Normal ECG:

The position of the heart relative to the electrodes are the important considerations in interpreting the configurations of the waves in each lead. The atria are located posteriorly in the chest. The ventricles form the base and anterior surface of the heart, and the right ventricle is anterolateral to the left. Atrial depolarization, ventricular depolarization, and ventricular repolarization move away from the exploring electrode a VR, therefore, P wave, QRS complex, and T wave are all negative (downward) defections. Leads aVL and aVF look at the ventricles and the deflections are therefore predominantly positive or biphasic **(Entmann et al., 1990).**

There is no Q wave in V_1 and V_2 and the initial portion of the QRS complex is a small upward deflection because ventricular depolarization first moves across the midportion of the septum from left to right toward the exploring electrode. The wave of excitation moves down the septum and into the left ventricle away from the exploring electrode, producing a large S wave. Finally , it moves back along the ventricular wall toward the electrode producing the return to the isoelectric line **(Foex, 1989)**.

Conversely, in the left ventricular leads (V_4-V_6) there may be an intial small Q wave (left to right septal depolarization), and there is a large R wave (septal and left ventricular depolarization) followed in V_4 and V_5 by a moderate S wave (late depolarization of the ventricular walls moving back toward the AV junction). There is considerable variation in the position 0 the normal hear, and the position affects the configuration of the electrocardiographic complexes in the various leads **(Nimmo et al., 1989)**.

The cardiac vector

Because the standard limb leads are records of the potential differences between two points, the deflection in each lead at any instant indicates the magnitude and direction of the axis of the lead of the electromotive force generated in the heart (cardiac vector). The vector at any

given moment in the two dimensions of the frontal plane can be calculated from any two standard limb leads (Figure 3). If it is assumed that the three electrode locations from the points of an equilateral triangle (Einthoven's triangle) and that the heart lies in the center of the triangle. These assumptions are not complenely warranted but calculated vectors are useful approximations **(Meduk et al., 1983)**.

An approximate mean **QRS** vector " electrical axis of the heart" is often plotted by using the average. This is a "mean vector" as opposed to an "instantaneous vector". The average QRS deflections should be measured by integrating the QRS complexes. However, they can be approximated by measuring the net differences between the positive and negative peaks of the QRS **(Meduk et al., 1983)**.

The normal direction of the mean QRS vector is generally said to be -30 to +110 degrees on the coordinate system (Figure 3). QRS vector is drawn as follows: perpendicular dropped from the midpoints of the sides of the equilateral triangle intersect at the center of electrical activity. In each lead, distances equal to the height of the R wave minus the height of the targestingative deflection in the QRS complex are measured off from the midpoint of the side of the triangle representing that lead. An arrow drawn from the center of electrical activity to the point of intersection of perpendiculars extended from the distances

measured off on the sides represents the magnitude and direction of the mean QRS vector **(Browdsky et al., 1997)**.

Left or right axis deviation is said to be present if the calculated axis falls to the left of -30 degrees or to the right of +110 degrees, respectively. Right axis deviation suggests right ventricular hypertrophy, and left axis deviation may be due to left ventricular hypertrophy, but there are better and more reliable electrocardiographic criteria for ventricular hypertrophy **(Foex, 1989)**.

Vectorcardiography

If the tops of the arrows representing all of the instantaneous cardiac vectors in the frontal plane during the cardiac cycle were connected, from first to last, the line connecting them forms a series of three loops: one for the **P** wave. one for the **QRS** complex, and one for the **T** wave. This can be done electronically and the loops are called vectorcardiograms, projected on the face of a cathode ray oscilloscope **(Nimmo et al., 1989)**.

His Bundle Electrogram

In patients with heart block, the electrical events in the **AV** node, bundle of His, and Purkinje system are frequently studied with a catheter containing an electrode at its tip that is passed through a vein to the right side of the

heart and manipulated into a position close to the tricuspid valve. Three or more standard electrocardiographic leads are recorded simultaneously. The record of the electrical activity obtained with the catheter is the His bundle electrogram (HBE). It normally shows an A deflection when the AV node is activated, an H spike during transmission through the His bundle, and a V deflection during ventricular depolarization **(Browdsky, 1997).**

With the HBS and standard electrocardiographic leads, it is possible accurately to time three intervals: (1) the PA interval the time from the first appearance of atrial depolarization to the A wave in the HBE which represents condction time from the SA node to the AV node; (2) the AH interval from the A wave to the start of the spike, which represents the AV nodal conduction time, and (3) the HV interval, the time from the H spike to the start of the QRS deflection in the ECG, which represents conduction in the bundle His and the bundle branches. The approximate values for these intervals in adults are PA 27 ms; AH 92 ms; and HV 43 ms These values illustrate the active slowness of conduction in the AV node **(Browdsky, 1997).**

Chapter III

Chapter III

PATHOPHYSIOLOGY OF PERIOPERATIVE ARRHYTHMIAS

Impulses normally arise from sinus node and are conducted via the specialized conducting system to the endocardial Purkinje network and myocardium. Any disturbance in this normal sequence of cardiac activation or deviation beyond accepted limits in rate or regularity of the heart beats is said to contribute to arrhythmias. Cardiac arrhythmias that originate in ectopic foci may be due to abnormal physiological mechanisms that are related to pathological changes in cardiac muscle. Arrhythmias may originate in the AV node, the His-Purkinje system or in atrial or ventricular muscle. In general, they are related to three different phenomena. These are:

1) **Enhanced automatcity in conducting tissues or myocardial cells.**
2) **Re-entry or reciprocating mechanisms in abnormal cardiac cells.**
3) **The occurrence of pathological after-potentials.**

These phenomena are believed to be responsible for the production of many tachyarrhythmias e.g. atrial flutter,

atrial fibrillation, ventricular extrasystoles, ventricular tachycardia and supraventricular tachycardias (including the wolff-parkinson- white syndrome) *(Nimmo et al., 1989).*

1) Enhanced automatcity in conducting tissues or myocardial cells

Automatic rhythm have been categorized as normal or abnormal according to the level of resting membrane potential from which they arise .

Normal automaticity aries from fully repolarized cells ($\geq$ - 90mv for pukinje fibres, $\leq$-75 mv for sinoatrial and nodal cells) which are subject to overdrive suppression.

Abnormal automaticity occurs in partially depolarized cells (range of membrane potentials -90 to -50) in which susceptibility to overdrive suppression decreases as resting membrane potential declines.

In a preparation with low potential, overdrive pacing rarely causes suppression which may result in post-overdrive enhancement of automaticity possibly due to release of endogenous catecholamines. Prolonged pacing may how ever cause some overdrive suppression *(Nimmo et al., 1989).*

Impule formation may also occur as a result of oscillations of membrane potential termed after depolarizations. After depolarization may be early that interrupts or delays normal repolarization. After depolarization may also be delayed following complete or near complete repolarization. Early after depolarization (EAD) occurs when cardiac tissue is exposed to catecholamines, drugs and to altered ionic conditions **(hypokalaemia, hypocalcemia) (Ming et . al 1994)**

- Delayed after depolarization (DAD) occurs in digitalis toxicity or cardiac tissue exposed to catecholamines in digitalis toxic Purkinje fibres is believed to be due to a transient inward current carried by Na^+ through a non specific membrane channel but in catecholamine superfused coronary sinus similar mechanism is uncertain **(Bigger, 1984)**

- Antiarhythmic drugs which shorten action potential duration might prevent triggered activity. While drug which prolong action duration might cause DAD. **(Hurst et, al, 1990).**

2) Re- entry and reciprocating mechanisms

The most common mechanism for tachyarrhythmias is reentry .four conditions are necessary to initiate and sustain reentry : two areas in the myocardium that differ in conductivity or refractoriness and that can form a closed electrical loop ; unidirectional block in one pathway (Figure 5 A-B);slow conduction of sufficient length in the circuit to allow recovery of the conduction block in the first pathway (Figure 5C); and excitation of the initially blocked pathway to complete the loop (Figure 5 D); reentry is usually precipitated by a premature cardiac impulse. *(Hurst et al., 1990).*

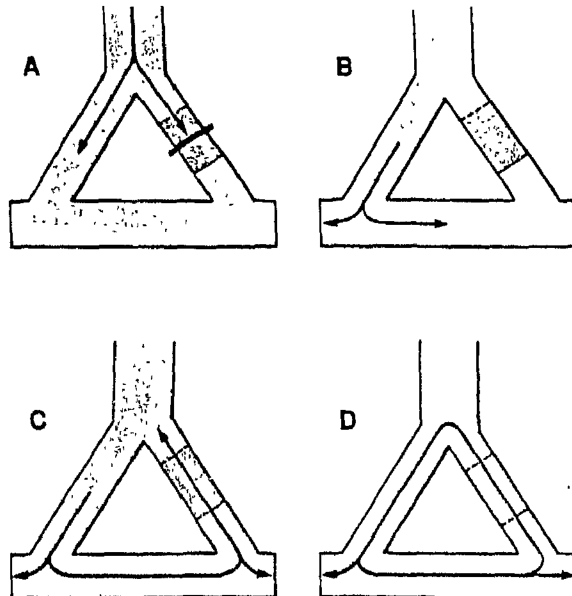


Fig. (5): The mechanism of reentry.

3. Pathological after-potentials

Pathological changes in ischaemic myocardium may cause the generation of spontaneous after-potentials after the action potential. These changes may be related to entry of calcium or sodium ions along ion-specific channels **(Zipes et al., 1995)**.

Causes of Perioperative Arrhythmias

Congenital

Many supra-ventricular arrhythmias arc congenital and most are benign. Accessory pathway tachycardias may be life-threatening. The significance of these arrhythmias lies in their ability to compromise hemodynamic stability; they almost never progress to ventricular arrhythmias. A congenital prolonged Q-T interval is seen at all ages and may predispose persons to ventricular tachyarhythmias **(Medak et al., 1983)**.

Acquired

Ventricular arrhythmias are seen commonly in association with ischemic heart disease, aortic stenosis, and other diseases associated with left ventricular hypertrophy. Their significance is equal to that of the underlying disease. Ventricular arrhythmias occurring immediately after an acute myocardial infarction or in the presence of congestive heart failure are dangerous in their own. This form probably represent ischemia-induced changes in excitability or refractoriness **(Andreali et al., 1987)**.

Atrial fibrillation is most often an acquired disease secondary to ischemic heart disease or related to aging. In patients with distended atria, such as mitral stenosis or chronic heart failure, atrial fibrillation is common and

adversely affect cardiac function. An acquired prolonged Q-T interval secondary to ischemic heart disease, electrolyte abnormality or commonly, drug side effects or toxicity may progress to polymorphic ventricular tachycardia (torsades de pointes). Central nervous system disease, such as closed head injury, intracranial hemorrhage, or stroke, is associated with all types of supraventricular tachycardia (SVT) and ventricular arrhythmias. The mechanism is not known, but if hemodynamic compromise is present, they must be treated primarily **(Andreoli et al., 1987)**.

Low potassium has long been implicated in the genesis of ventricular arrhythmias. Acute loss of potassium may trigger dangerous ventricular arrhythmias and should be treated in any setting. Abnormal levels of sodium are not implicated in arrhythmias; however, low magnesium levels may interfere with sodium-potassium pump and produce primarily SVT. In cardiac surgery, the administration of 2g magnesium sulfate, regardless the magnesium level, has been shown to reduce the incidence of postpump SVT. Acute changes in *p*H are associated with alterations in the transmembrane potassium gradient and available ionized calcium. Nevertheless, arrhythmias are not a major sequelae of acute *p*H changes **(England et al., 1992)**.

Anesthetics

For the most part, all anesthetics have calcium antagonistic properties and are antiarrhythmic. Halothane as a inhalational anaesthetic is widely used, independently sensitize the heart to circulating endogenous or exogenous catecholamines. The mechanism of halothane's effect is unclear but may relate to decreased gradient between the peak negative potential and threshold potential of the cell coupled with its direct slowing of the rate of spontaneous diastolic depolarization. Drugs that release catecholamines or block their reuptake will potentiate this effect of halothane **(Hirsch et al., 1988)**.

Chapter IV

Chapter IV

DIAGNOSIS OF PERI-OPERATIVE ARRHYTHMIAS

All monitors currently used require the interpretive skills of the anesthetist for the control of anaesthesia with the prime objective of ensuring the safety of the patient. The overall functions of any monitor are: to aid the anaesthetist in the maintenance of the patient"s physiological homeostasis, to detect changes in the rhythm of the patient to a dangerous one , and to warn of changes in the function of the anesthetic equipment which may be hazardious on the patient under anaesthesia. Intra-operative monitors include, cardiovascular monitoring respiratory monitoring temperature, and the extent of neuromuscular blockade **(Bedford, 1993)**.

CARDIOVASCULAR MONITORING

These include: electrocardiography, central pressure monitors, cardiac output, blood pressure, transesophageal echocardiography and recent electrocardiographic techniques.

1. Electrocardiography :

All patients undergoing anesthesia (and surgery) deserve E.C.G. monitoring. Although the E.C.G. says little concerning about the adequacy of pump function or the safety of anesthetic technique, it facilitates detection of both ischaemia and rhythm disturbances, with proper lead placement, even the standard three-lead monitors currently available can achieve these goals **(Atlee et al., 1990).**

I. Indications:

a. Diagnosis of ischaemia.
b. Diagnosis of dysrhythmias.
c. Diagnosis of conduction defects.
d. Diagnosis of electrolyte disturbances.

II. Techniques

a. The three electrode system. This system utilizes only electrodes on the right arm, left arm and left leg. One pair of electrodes can be selected for monitoring at one time; three ECG leads (I, II, III) can therefore be examined (**Bazaral et al., 1981).**

b. The five-electrode system: The use of five electrodes (one lead on each extremity and one precondial lead) allows the recoding of the six standard frontal limb leads as well as on precordial unipolar lead. The unipolar lead is usually placed in the V5 position,

along the anterior axillary line in the fifth intercostal space.

1. Advantage: with the addition of only two electrodes to the ECG system, seven different leads can be monitored simultanealy. More important all but the posterior wall of the myocardium can be monitored for ischaemia. Multiple ECG leads will also be useful in the diagnosis of atrial versus ventricular dysrhythmias.

2. Disadvantage: The V5 electrode should not interfere with operative field for a median sternotomy, although it will certainly interfere with a left thoracotomy incision **(Atlee et al., 1990).**

C. Invasive ECG

1. Esophageal: esophageal leads can be incorporated into the esophageal stethoscope and are very useful for the diagnosis of atrial dysrhythmias. 100% of the atrial dysrhythmias were correctly diagnosed with the esophageal leads. Lead II led to a correct diagnosis in 54% of cases and V5 in 42% of cases **(Cyran et al., 1989).**

2. Endotracheal: ECG leads have been incorporated into endotracheal tube, although such endotrached tubes are presently not available commerically, they may be

usefull in pediatric cardiac patients for the diagnosis of atrial arrhythmias **(Smith et al., 1985).**

C. Multipurpose pulmonary artery (PA) catheter

The multipurpose PA catheter has all the features of a standared PA catheter. In addition, three atrial and two ventricular electrodes have been incorporated into the catheter. These electrodes allow not only recording of intracavitary ECGs but also atrial and atrioventricular pacing **(Trankina et al., 1989).**

D. Epicardial electrodes

It is common practice for cardiac surgeons to place ventricular and atrial epicardial pacing wires prior to weaning the patient from cardiopulmonary bypass, or before sternal closure.

Although the primary intent of these wires is to allow atrioventricular pacing in the post bypass periods they can alse be utilized to record atrial and ventricular epicardial ECGs. These leads are most useful in the post operative diagnosis of complex conduction problems **(Trankina et al., 1989).**

III. Recording and interpretation

To fully utilize the capabilities of ECG monitoring, particular attention must be paid to elimination of major sources of antifact and error.

a. Patient-electrode interface: The electrical signal generated by the heart and monitored by the ECG is very weak, amounting to only 0.5-2.0 mv at the skin surface.

- The skin should be clean and free of all dirt.
- Skin resistance can be as great as 1.000.000 (ohms).
- To avoid problem of muscle artifact, electrodes should be placed over bony prominences when even possible.

B. Electrodes: Electrodes should be of the silver chloride type to avoid resistance mismatch between various kinds of electordes.

C. Leads and connecting cables:

1. Inserlation, the main source of artifact from ECG leads is loss of integrity of the insulation on leads and connecting cables.

2. Motion artifact, lead movement will lead to artifact.

3. Crossing cables, crossing other monitoring cables over the ECG leads will cause significant interference.

d. Electronic filtering system, most ECG monitors have filteres to decrease environmental artifacts. They can usually operate in two modes.

1. Monitoring mode (0.5-40 Hz) the monitoring mode eliminates both low and high-frequency artifacts.
2. Diagnostic mode (0.05-100 Hz) this mode does not filter the higher frequency signals but is more subjected to artifact.

(Cyran et al., 1989).

IV. Recommendations for ECG monitoring:

It is recommended using a five-electrode surface ECG monitor during anesthesia. This monitor should be able to display at least two leads simultaneously. The use of two simultaneously leads to monitor two different areas of myocardium supplied by two different coronary arteries facilitates the diagnosis of dysrhythmias **(Smith et al., 1985).**

2. Arterial blood pressure :

Arterial blood pressure can be measured using either indirect or direct methods .

A. Indirect measurement :

This can be done either using a Riva- Rocci occlusive cuff with auscultation of the Korotkoff sounds or through a wide range of instruments such as the Dinamap or Accutorr, which are automatic electronic extensions of the oscillotonometer **(Bedford, 1993).**

B. Intra–arterial pressure monitoring

Intra-arterial blood pressure monitoring is indicated in:

- Severe poorly controlled hypertension, cardiac disease, unstable angina or recent myocardial infarction .
- Cerebral vascular disease : transient ischaemic attacks or recent stroke .
- Operations: Cardiac surgery, major surgery on the aorta, thoracic aorta surgery, carotid surgery, cerebral aneurysm surgery.
- Pulmonary disease: Severe pulmonary disease requiring blood gas measurement, one-lung ventilation, pulmonary hypertension .
- Others: inability to measure pressure non-invasively, sepsis and controlled hypotensive techniques.

(Bedford , 1993).

3. Central venous pressure monitoring (C.V.P.):

The C.V.P. is the pressure in the right atrium and most accurately reflects alteration in the volume or compliance of the right atrium or the right ventricle ; tricuspid or pulmonary valvular dysfunction or the effect of increased right ventriclar afterload (pulmonary hypertenion) **(Cardner and Hollingsworth, 1993).**

Indications for central venous pressure monitoring

- Assessment of blood volume :
  Hypovolemic shock .
  Septic shock .

- Assessment of right ventricular dysfunction associated with:
  Parenchymal pulmonary disease .
  Pulmonary hypertension .
  Acute right ventricular infarction.
  Cardiac tamponade .
- When pulmonary artery catheterization is risky or technically diffcult :
  Tricuspid insufficiency .
  Serious ventricular dysrhythmias .
  Left bundle branch block .
  Pulmonary stenosis .
- Others:
  Sitting craniotomy.
  Difficult venous access.

(Nunn et al., 1989)

4. Pulmonary vascular pressure monitoring :

In 1970, Swan et al., introduced a multilumen, balloon-tipped, radio-opaque, polyinyl catheter which reliably passed into the pulmonary artery via the right heart by blind floatation without fluoroscopy. Pulmonary artery monitoring enables measurement of central venous pressure, pulmonary artery pressure (systolic, diastolic, mean), pulmonary capillary wedge pressure, cardiac output and mixed venous O_2 sturation. Specialized catheters can be used to measure right ventricular pressures, ejection

fraction and to allow temporary cadiac pacing **(Mangano, 1993)**.

Indications for use of pulmonary artery monitoring during major surgery

A. Ischaemic heart disease , with :

1. Acute myocardial infarction .
2. History of ventricular failure .
3. Cardiomegaly .
4. Ejection fraction less than 0.50.

B. Severe valvular heart disease .

C. Significant pulmonary disease, with

1. Respiratory failure and pulmonary oedema .
2. Pulmonary embolism .
3. Pulmonary hypertension .

D. Shock requiring massive volume , inotropes or vasoactive agents .

E. Aortic surgery with supracoeliac or more proximal clamping of the aorta .

F. Cardiac surgery :

1. Coronary artery bypass grafting .
   i) Ejection fraction less than 0.40.
   ii) Recent myocardial infarction .
   iii) Unstable angina .
   iv) Intercurrent valvular heart disease .

2. Valvular replacement .
3. Others:
   i) Congenital heart disease with pulmonary hypertension .
   ii) Idiopathic hypertrophic subaortic stenosis .
   iii) Pericardiotomy with tamponade .

(Mangano, 1993).

5. Trans-oesophageal echocardiography (TEE):

TEE provides assessment of (1) the ischaemic state via detection of regional wall motion abnormalities and wall thickness changes (2) ventricular function via measurement of estimated ejection fraction , wall shortening and ventricular volumes; and (3) others such as valvular function, intracardiac air and intracardiac masses . In addition, new advances in the development of TEE include the addition of Doppler colour flow and contrast echocardiography **(Schuster and Nada, 1985).**

The principle of echocardiography is based on reflected sound waves from the surfaces of internal organs. "Ultrasound" frequencies used in echocardiography are in a range from 1 to 7 MHz. The TEE system conists of an ultrasonograph system and echoprobe. The ultrasonograph contains signal processing software which allows transmission, detection of ultrasonic pulses and display of the images in both the M mode and two dimensional

format. The probe is usually placed in the oesophgus after the patient's trachea has been intubated. Following entry into the oesophagus, it is advanced to obtain several sectional views of the heart **(Smith et al., 1995)**

Ischaemic myocardium develops wall motion electrocardiographic abnormalities (systolic dysfunction then diastotic dysfunction) that often preceeds ST segment changes and may offer a more sensitive means of detecting myocardial ischaemia. Hence TEE may be a more sensitive clinical monitor than ECG, and maybe a better predictor of cardiac morbidity **(Topol et al., 1996).**

6. Recent diagnostic and monitoring ECG:

These include, body surface mapping direct cardiac mapping and signal averaging techniques.

- Body surface mapping:

Body surface mapping has been used to localize the size of myocardial ischaemia, localization of ectopic foci or accessory pathways and also to differentiate aberrant supraventricular tachycardia from ventricular tachycardia **(Pogwizd,1995).**

- Direct cadiac mapping:

This is done via catheter electrodes at the time of surgery to identify and localize the site of rhythm

disturbance. Mostly used during cardiac surgery for electrical or surgical ablation **(Pogwizd, 1995)**.

- Signal averaging techniques:

This has been applied clinically most often to detect late ventricular potentials of 1-25 V. Late potentials have been recorded in patients with ventricular tachycardia (not related to ischaemia) as in cardiomyopathies **(Pogwizd, 1995).**

RESPIRATORY SYSTEM MONITORING:

Pulmonary ventilatory troubles or disturbances in the composition of respired gases may be the causal factor for cardiac dysrhythmias; on the other hand, cardiac dysrhthmias resulting in major changes in respiratory functions indicate malignant ectopics. The major parameters to be monitored here are: monitoring of pulmonary ventilation, airway pressure analysis of composition of respired gases, capnography and pulse oximetry **(Bushman, 1994).**

1. Pulmonary ventilation:

Pulmonary ventilation monitoring is to monitor the movement of air in and out of the lungs. This can be done through the auscultation of breath sounds or watching the movement of the chest wall. However, impedance plethysmography can give a good indication of the changes

in ventilation. This method extracts the signal from ECG leads. As air moves in and out of the chest, the resistance between the ECG electrodes will change and this is sensed as a change in voltage across the high impedance current source **(Conway, 1990).**

2. Airway pressure:

This is easily measured with either an aneroid gauge or an electric manometer. Airway pressure warn a number of potentially life-threatening situations. In the spontaneoualy breathing patient. It warn if the expiratory valve or the scavenging line becomes obstructed, either of which may result in barotrauma of the lung which in turn result in unexpected frequent ectopics or constant dysrhysmias **(Conway, 1990)**.

More importantly, in ventilated patients, it gives an indication that the pressure are within the expected limits for the patients with respect to the measured tidal volume. Airway pressure gauges are also useful indication of the application of positive end expiratory pressure (PEEP) and the surest and fastest method of detecting a disconnection of the patients circuit **(Coriat et al., 1995).**

3. Analysis of composition of respired gases:

Monitoring of the composition of the respired gases comprises oxygen concentration in the inspired gas, infrared analysis of respired carbon dioxide (capnography),

anaesthetic gases, vapour and nitrogen. However of common use are the capnography and pulse oximetry **(Coriat et al., 1995).**

4. Capnography:

Capnography is a routine anaesthetic monitor. It yields a great deal of information in addition to the alveolar PCO_2.. Gas can be continuosly sampled from the patient's airway and drawn through a cuvette where the carbon dioxide concentration is measured by infrared absorption. The end-tidal CO_2 ($ETPCO_2$) is a useful measure of the adequacy of ventilation during anaesthesia. There is a mean arterial / end - tidal PCO_2 gradient of 5 mmHg. A sudden decrease in $ETPCO_2$ may be due to apnea or due to cardiac arrest which stops the transport of carbon dioxide to the lungs. The capnograph is a useful monitor to detect air embolism as well as an indicator of correct placement of tracheal tube **(Cormack and Powell, 1997).**

5. Pulse oximetry:

Absorbance of the light as it passes through the finger is determined by 2 factors. The first is a static component which involves the thickness of the finger, the amount of bone and the pigmentation of the skin. The second is a pulsatile component and is due to the blood pulsed through the finger. Pulse oximetry reflects the instantaneous oxygenation of the blood with change in inspired oxygen concentration **(Bushman, 1994).**

MONITORING OF TEMPERATURE

Hypothermia is very common following anaesthesia and surgery. The air conditioned environment, open cavities and the administration of cold blood or fluids are all some of the causes of hypothermia. Atrioventricular conducation disturbances, atrial fibrillation and ventricular extrasystoles are seen when low temperatures are reached. A common cause of death is ventricular fibrillation or cardiac arrest, when temperature below 29 C is reached. Temperature monitoring is usually carried out in the surgical patient using thermistor or thermocouples **(Waller et al., 1990).**

Other monitors which may be facultatively used in certain surgical procedures or particular types of dysrhythmia patients include neuromuscular junction monitors, evoked potentials for the depth of anaesthesia with cerebral function monitors and electrolyte level in blood **(Waller et al., 1990)**.

Chapter V

Chapter V

MANAGEMENT OF PERIOPERATIVE ARRHYTHMIAS

The perioperative management of dysrhythmia cases needs a paper understanding of the type of arrhythmia and its mechanism. In emergencies action often needs to be taken by the most jonior doctor in the team and some rote recommendations are then necessary. However the diagnosis and elective treatment of chronic or episodic dysrhythmias require greater skill to ensure that the risk-benefit equation receives the correct solution. As will become clear antidysrhythmis drugs have a hard time proving superior safety or efficacy over use of the defibrillator and pacing wire.

(I) ANTIARRHYTHMIC DRUGS

The goal of arrhythmic therapy may be to restore normal sinus rhythm, abolish ectopic beats, or control heartrate. Antiarrhythmic agents are currently divided into four major classes based on their mechanism of action.

Classification of Drugs

Class 1: soduim channel blockade. These drugs restrict the rapid inflow of sodium during phase 0 and thus slow the maximum rate of depolarization. Another term for this proparty is membrane stablizing activity. These drugs may be subclassified as follw.

A- Drugs that lenghens action potential duration and refractoriness (eg.quinidine ,disopyramide , procainamide)

B-Drugs that shorten action potential duration and refractoriness (eg lignocaine , tocainide , phenytoin)

C-Drugs that have negligible effect on action potential duration and refractoriness (eg.flarcanide , propafenone.)

Class II: Catecholamine blockade. Propranolol and other beta adrenoceptor antagonists reduce background sympathetic tone in the heart reduce automatic discharge (phase 4) and protect against adrenergically stimulated ectopic pacemakers.

Class III: lengthening of refactoriness (without effect on sodium inflow in phase 0.)prolongation of cellular refractoriness (phase 1, 2, 3) beyond a critical point may prevent a reentry circuit being completed (eg amiodarone , bretyluim also sotalol).

Class IV: calcuim channel blockade, these drugs depress the slow inward calcuim current (phase 2) and prolong condction and refractoriness particularly in S A and A V nodes which may explain their effectiveness in terminating paroxysmal supraventricular tachycardia (e.g. verapamil) **(Roden, 1994)**.

Principal drugs

1. Procainamide:

Mechanism of action:

Procainamide depresses automaticity by decreasing the slope of phase 4 depolarization. By increasing refractoriness, procainamide can prevent re-entry by converting a unidirectional to a bidirectional block.

In addition, it exhibits weak autonomic ganglionic blocking action which impairs cardiovascular reflexes.

Indication:

Supraventricular (SVT) and ventricular arrhythmias. (premature ventricular contractions (PVCs) and ventricular tachycardia (VT).

Pharmacokinetics:

The distribution half-life is <10 minutes after an intravenous dose. About 20 percent of the drug is bound to serum proteins.

Approximately half the dose is eliminated unchanged by renal excretion and half by hepatic metabolism with an overall elimination half-life of about 4 hours. It should be remembered that patients with renal failure will convert

nearly all the parent drug via hepatic metabolism to N-acetylprocainamide (NAPA), which is excreted entirely through the kidneys and will therefore attain toxic levels of procainamide if administered chronically.

Dosage and administration:

Slow injection with careful haemodynamic and ECG monitoring is essential. Rapid administration can result in severe hypotension from both myocardial depression and peripheral vasodilatation. A dose of 100 mg IV should be given slowly over 1 minute and repeated every 5 minutes until the arrhythmia is controlled, which often occurs with a total dose of 5 to 15 mg/kg.

Once the arrhythmia has been controlled, a continuous infusion should be started at 1 to 4 mg/min. With renal disease, the loading dose is unchanged but the maintenance dose should be decreased.

If the patient has advanced renal disease, one must consider switching to quinidine, for maintenance therapy.

Toxicity:

Myocardial depression, hypotension, QRS complex and QT interval prolongation, heart block and ventricular ectopy are the major acute toxic effects.

Procainamide directly slows the atrial rate in atrial flutter or fibrillation, while conduction through the A-V node may be increased by vagolytic effects. This may result in a paradoxical increase in ventricular response. Adequate digitalization reduces but doesn't abolish this danger **(Wellens and Brugada, 1995).**

2. Lidocaine:

Mechanism of action:

In therapeutic concentrations, lidocaine's major effect is to decrease the slope of phase 4 depolarization in Purkinje fibres hence reducing automaticity.

Indications:

Lidocaine is the drug of choice for ventricular arrhythmias but ineffective against supraventricular arrhythmias.

Pharmacokinetics:

After intravenous injection, the drug distributes rapidly, with a distribution half-life of < 10 minutes. About

60 percent of lidocaine in plasma is bound to albumin. It's primarily (95 percent) metabolized in the liver, with an elimination half-life of 2 to 3 hours The apparent volume of distribution is decreased and elimination half life increased in congestive heart failure, liver disease, or shock.

Dosage and administration:

Lidocaine is administered as an intravenous bolus of 1 to 1.5 mg/kg. Injections should be given over 5 to 8 minutes as necessary to a total of 3 mg/kg. When there is a therapeutic response, an infusion of 1 to 4 mg/min should be started to maintain an effective concentration.

If there is no response to bolus injections, a switch should be made to another drug. One must remember to use smaller doses for elderly patients and those patients in heart failure or shock.

Toxicity :

Is primarily limited to central nervous system (CNS) disturbances, ranging From CNS stimulation (muscle twitching, disorientation, excitement and convulsions) as plasma levels increase CNS depression occurs (progressive drowsiness). Lidocaine doesn't produce adverse cardiovascular effects, although it can depress ventricular performance in patients with pre-existing severe left ventricular dysfunction. In addition, it can rarely cause

further slowing of the heart rate in patients with sinus bradycardia **(Wellens and Brugada, 1995).**

3. Phenytoin (Dilantin):

Mechanism of action:

This drug depresses phase 4 diastolic depolarization in a manner similar to lidocaine. It is also effective in abolishing activity triggered by digitalis-induced after depolarizations in cardiac Purkinje fibres, which may explain its efficacy against certain rhythms due to digitalis toxicity.

Indications:

Phenytoin is sometimes useful against digitalis-induced ventricular tachycardia or paroxysmal atrial tachycardia with A-V block.

Pharmacokinetics:

After intravenous injection, the distribution half-life is about 15 minutes, somewhat slower than that of procainamide and lidocaine. The drug is approximately 85 percent bound to serum proteins, mainly albumin, and therefore a greater fraction is unbound in hypo-albuminemic patients. The liver metabolizes nearly 95 percent of the drug; however its metabolism is relatively slow with elimination half-life of about 24 hours. Its

elimination is not substantially altered by changes in hepatic blood flow.

Dosage and administration:

Intermittent intravenous administration through a central venous line is recommended. Peripheral administration can cause severe phlebitis because the solution is highly alkaline. This drug must be given only in normal saline solutions. Dosage is 100 mg every 5 minutes until the arrhythmia can be controlled. Doses above 1000 mg are rarely required and can lead to toxicity. Infusions are not used because of the long half-life and difficulties with intravenous administration.

Toxicity:

Rapid administration has been associated with respiratory arrest, severe hypotension, ventricular ectopy, and death. Other major toxic effects include drowsiness, nystagmus, nausea, vertigo, and other cerebellar signs. These latter signs will be masked under anaesthesia **(Zipes, 1997).**

4. Propranolol

Mechanism of action:

The antiarrhythmic properties of propranolol result principally from inhibition of β-adrenergic stimulation of

the heart. Sympathetic stimulation increases automaticity by increasing phase 4 depolarization, enhances conduction velocity, and shortens the refractory period especially in supraventricular tissues. Propranolol, by B-adrenergic blockade, leads to slowing of the SA node rate, prolonged AV nodal conduction, and refractoriness.

Indications:

Propranolol is used for control of supraventricular tachycardia (SVT), and slowing ventricular response in artial fibrillation and flutter. It is occasionally effective for ventricular arrhythmias that do not respond to conventional measures,

Pharmacokinetics:

I.V. administration eliminates the extensive "first-pass" hepatic uptake and degradation that occur after oral administration.

Therefore, small I.V. doses can be very effective. The elimination half-life is about 2 to 3 hours after intravenous administration mainly because of hepatic metabolism (95 percent). Approximately 90 percent of this drug in plasma is bound to serum proteins. Its elimination is significantly reduced when hepatic blood flow decreases, as in congestive heart failure or shock.

Dosage and administration:

I.V. administration requires careful titration, with monitoring of heart rate, blood pressure, and occasionally, left ventricular filling pressure. The drug should be given in 0.5 to 1.0 mg increments over 2 to 5 minutes up to a total dose of 0.1 mg/kg. Smaller doses (0.5 to 1 mg) are often effective for SVT or for controlling ventricular response in atrial fibrillation or flutter.

Toxicity:

Is primarily due to B-adrenergic blockade. Profound bradycardia or asystole may occur, especially in patients with sick sinus syndrome. Acute left ventricular failure may be precipitated in patient with pre-existing left ventricular dysfunction. Acute bronchospasm may occur in patients who are asthmatic or who have chronic bronchitis secondary to blockade of B-receptors in the lung . **(Wellens and Brugada, 1995).**

5. Bretylium:

Mechanism of action:

Bretylium, an adrenergic neuronal blocking agent, increases the threshold for ventricular fibrillation in both normal and ischaemic myocardium. The importance of its

adrenergic neuronal blocking activity in treatment of cardiac arrhythmias and ventricular fibrillation remains to be established.

Indications:

This drug is indicated for treatment of life threatening ventricular arrhythmias, principally ventricular tachycardia and fibrillation that have not responded to adequate doses of antiarrhythmic agents such as lidocaine or procainamide.

Pharmacokinetics:

Elimination is most entirely by renal excretion without significant metabolism. The elimination half life is about 6 to 10 hours with normal renal function.

Dosage and administration:

A dose of 5 to 10 mg/kg is infused over 10 to 20 minutes after diluting 2 ampoule (500 mg in 10 ml of water) to a volume of 50 ml or more In extreme emergencies (i.e. during cardiac resuscitation) a dose of 5 mg/kg can be injected undiluted as a bolus, which can be repeated in a dose of 5 to 10 mg/kg every 15 to 30 minutes up to a total dose of 30 mg/kg. Once ventricular ectopy has been controlled, an additional 5 to 10 mg/kg may be given every 6 to 8 hours, or alternatively, an infusion of 1 to 2 mg/min may be started.

Toxicity:

Hypotension is the most consistent Adverse action owing to the blocking effects on the sympathetic nervous system; often there is a substantial orthostatic component.

Mild hypertension may transiently be seen as a result of the initial release of norepinephrine from nerve terminals. Nausea and vomiting are occasionally after intravenous administration, especially rapid administration **(Zipes, 1997).**

6. Verapamil:

Mechanism of action:

The calcium channel blockers selectively block slow channels by inhibiting the normal calcium ions influx into cells. Within the conduction system, verapamil's slow channel activity is most important on the SA and AV nodes, where it prolongs- AV nodal conduction and refractoriness and depresses the SA node discharge It has little effect on the His-Purkinje system.

Decreases in systemic vascular resistance (SVR) and blood pressure are directly related to its calcium channel blocking activity in systemic arterioles. Depression of myocardial contractility occurs as a result of interference

with calcium-mediated excitation-contraction coupling.

Indications:

Verapamil is useful in the treatment of supraventricular tachycardias. It's very effective in slowing ventricular rate in atrial flutter and fibrillation. Intravenous infusion of diltiazem is the newest approach to rate control of atrial flutter or fibrillation.

Pharmacokinetics:

Verapamil is highly protein-bound, and the presence of other highly protein-bound drugs
lidocaine, or propranolol can significantly
(active) fraction. Its mean distribution half-life is only
minutes, and its clinical duration of action after an intravenous dose is only 10 to 20 minutes.

Metabolism is through the liver with an elimination half-life of 2 to 7 hours, although, this is prolonged in patients with liver disease.

Dosage and administration:

Intraoperatively, it is recommended to start a dose of 2.5 mg and repeat as necessary to a dose of 15 mg.

Toxicity:

Hypotension is a major side effect, although

bradycardia, asystole, AV block have been noted, usually in patients with pre-existing conduction disease or sick sinus syndrome. Myocardial depression is uncommon in patients with reasonable left ventricular function.

Caution is advised in patients taking B-blockers and those with severe left ventricular dysfunction **(Wellens and Brugada, 1995)**.

7. Adenosine:

Mechanism of action:

Adenosine is an endogenous nucleotide. In supraventricular tissues it increases potassium conductance, which results in shortening the duration of action potential, hyperpolarization, slowing of firing of SA nodal cells, and in depression of the action potential in the AV node. These effects account for the ability of adenosine to terminate certain types of SVT.

Indication:

Adenosine has been approved for use in the treatment of SVT. Specifically, it is effective for reentrant tachycardias that use the AV node as part of the re-entrant circuit (e.g., AV nodal re-entry and AV reciprocating tachycardia). For arrhythmias such atrial flutter and fibrillation, it causes only transient AV block, and if given

for sinus tachycardia, it will also result in slowing of the SA node.

Adenosine also has utility as a diagnostic tool. For example, in patients with wide complex tachycardia, termination with adenosine suggests SVT with aberrancy as the mechanism. The only kind of ventricular tachycardia that responds to adenosine is a rare type that is due to catecholamine triggered activity.

Since adenosine may sometimes precipitate transient atrial flutter or atrial fibrillation, caution should be exercised when administering adenosine to patients with manifest pre-excitation. The onset of atrial fibrillation could result in rapid anterograde conduction over an accessory pathway.

Pharmacokinetics:

Adenosine has a very brief half-life, less than 1.5 seconds. Its inactivation occurs by cellular uptake. In the cell it is either deaminated to inosine or phosphorylated to adenosine monophosphate (AMP).

The actions of adenosine are potentiated by nucleoside transport blockers such as dipyridamole and are attenuated by adenosine antagonists such as methylxanthine derivatives.

Dosage and administration:

Adenosine is only available as an intravenous agent. It should be delivered as a rapid bolus, followed by a saline flush. Adenosine's effects are apparent within 10 to 20 seconds when it is administered through a central line.

The haemodynamic response to a bolus injection is minimal. The usual adult starting dose is 6 mg and is followed by 12 mg if the initial dose proves ineffective. Children should receive incremental doses beginning with 50 μg/kg.

Patients taking theophylline, because antagonist properties, may not respond at adenosine.

Toxicity:

The most common side effects are facial flushing, dyspnea and chest pressure. These symptoms subside in less than 60 seconds. Adenosine may exacerbate bronchoconstriction in asthmatic patients and therefore alternative therapy may be prudent **(Zipes, 1997).**

8. Digoxin:

Mechanism of action:

Digoxin reduces the ventricular rate in atrial fibrillation by directly prolonging the effective refractory

period in the AV node and also indirectly by increasing vagal activity and reducing sympathetic activity. Ventricular rates are easier to control during fibrillation than during flutter.

Indications:

As an antiarrhythmic, digoxin is indicated for rate control of atrial fibrillation, atrial flutter, and supraventricular tachycardia.

Pharmacokinetics:

After intravenous administration, the time to onset of action is 20 to 30 minutes, with maximal action achieved within 2 hours. Digoxin is about 25 percent bound to plasma albumin. Elimination is mainly by glomerular filtration in the kidneys. The elimination half-life is approximately 36 hours, although this is prolonged in patients with reduced renal function.

Dosage and administration:

The intravenous route is preferred when digoxin must be given parenterally. In adult patients, the initial intravenous loading dose of 1 mg to 1.5 mg given in increments are unlikely to cause toxicity and can be supplemented by further increments if indicated.

Toxicity:

Alteration in cardiac rate and rhythm may stimulate almost every known rhythm disturbance. The most frequent arrhythmias with digoxin overdose are PVCs, either unifocal or multifocal often coupled as bigeminy or trigeminy. Other common arrhythmias include AV junctional escape rhythms (regularization of rate in artial fibrillation), non paroxysmal junctional tachycardia, paroxysmal atrial tachycardia with A-V block, second degree A-V block, ventricular tachycardia and fibrillation. Cardiac toxicity is enhanced in the hypokalemic patient **(Zipes, 1988).**

(II) Electrical Treatment of Arrhythmias

This includes pacing and direct current shock.

A. Pacing:

While drug therapy is an effective means of treating arrhythmias, there are difficulties with side effects and potential cardiovascular toxicity. Pacing has no such toxicity and in addition is immediate in its onset and termination, easily controllable, convenient, and safe **(Zaidan, 1993).**

Mechanisms of pacemaker action:

a. Overdrive pacing to prevent arrhythmias:

The atrium or ventricle is paced at a rate faster than the spontaneous rate. This may be useful in suppressing tachyarrhythmias of any type as well as PVCs.

b. Rapid atrial pacing to terminate tachyarrhythmias:

- The atrium is paced either via wires sutured directly to the heart at the time of surgery or from a pacing catheter.
- A pacing generator is needed that will generate impulses as fast as 500 beats per minute (bpm).
- Used for treatment of SVT where the atria are r 20 bpm faster than spontaneous rate. After ca achieved, the pacing is abruptly stopped. If tl the procedure is repeated at faster rates in in of 10 bpm.

  The technique is unlikely to be effective beyond 100 bpm above the tachycardia rate, and it may precipitate atrial fibrillation.

- Also can be used for treatment of atrial flutter:

  The procedure used here is referred to as ramp atrial

pacing. Pacing at a continuously increasing rate for 5 to 20 seconds is sometimes effective for termination of atrial flutter. The low-rate end of the ramp is the flutter rate; the high rate ranges from 50 to 125 bpm above the flutter rate. The faster the atrial pacing rate, the greater the risk of converting the rhythm to atrial fibrillation. It must be strongly emphasized that rapid pacing should be attempted only by personnel experienced in this technique **(Zaidan, 1993)**.

c. Rapid ventricular pacing:

This technique may sometimes be used to terminate VT and even SVT. There is a risk of precipitating a more rapid VT or VF The inherent risk of this technique restrict its use only to experienced electrophysiologists.

Cardiovascular effects of pacing:

(a) Ventricular pacing increases cardiac output simply by increasing the heart rate to a physiologic level. A reasonable initial rate occurs between 75 to 80 bpm. Ventricular pacing, however doesn't preserve the atrial kick, which is normally responsible for about 20 percent of the cardiac output.

(b) Atrial pacing increases the cardiac output not only by increasing the heart rate but also by preserving the atrial kick. High atrial pacing rates in the presence of conduction disease lead to A-V block. If second-

degree block occurs when the patient is being atrially paced, the pacing rate should be slowed or A-V sequential pacing is instituted

(c) A-V sequential pacing should be used when the patient's atrium is not contracting but can contract when electrically stimulated or when the atrial and ventricular contractions are dissociated. An example is a junctional rhythm associated with adequate A-V conduction, Occasionally, shortening the PR interval will increase the cardiac output in a patient with first-degree block Sequential pacing is indicated in patients with complete heart block but not in patients with atrial fibrillation. The pacing pulmonary artery catheter can be used to establish AV pacing. The correct PR interval is determined by performing cardiac outputs at different PR intervals (e.g., at 200, 175, 150, and 120 ms) while holding the heart rate constant. Generally, a thick ventricle require a long PR interval to provide more time for ventricular filling. The highest cardiac output determines the correct PR interval for that patient **(Baig and Perrins, 1991).**

Nomenclature of pace-makers:

A five-letter generic code is used to classify pace-makers. The first three letters refer to antibradycardic functions, the last two relate to programmability and rate of arrhythmia control.

(a) First letter - chamber(s) paced.

1. A = atrium.
2. V = ventricle.
3. D = both.

(b) Second letter = chamber(s) sensed.

1. A = atrium.
2. V = ventricle.
3. D = both.
4. O = non.

(c) Third letter = response.

1. T = triggered: the sensed wave initiates an impulse from the pace-maker, which occurs during the refractory period.
2. I = inhibited: The sensed wave turns the pace-maker off.
3. D = both (T and I).
4. 0 = none.

(d) Fourth letter = Programmable functions.

1. P = programmable for rate and/or output.
2. M = multiprogrammable (i.e., for more than rate and output).
3. C = communicating.
4. R = rate modulation.

(e) Fifth letter = antitachyarrhythmic functions.

1. O= none.

2. P = pacing.
3. S = shock.
4. D= both.

(Kelly and Royster, 1989)

Types of pace-maker generators:

(a) Asynchronous:

This type of pace-maker has no sensing circuit to detect intrinsic R waves. The asynchronous pacemaker will exhibit competition with the patient's heart rate so that the ECG will show normal QRS complexes, paced beats, non effective pacing impulses in the QRS or T waves, and also fusion and pseudo-fusion beats.

(b) Synchronous:

Since this type of pace-maker includes a circuit detects intrinsic depolarizations, synchronous gene eliminate the problem with competition. One of two e will occur when the patient's **R** wave is detected, the maker will be either activated or inhibited **(Kelly Royster, 1989**.

Preoperative evaluation of the patient w pacemaker:

(a) Complete evaluation of the patient with empha understanding the physiology, severity,

progression of the disease process that led to pace-maker insertion:

1. A high incidence of coronary artery disease, hypertension, diabetes and peripheral vascular disease should be expected.

2. Usually patients should receive their prescribed doses of medications such as B-blocking and calcium channel blocking drugs. Antiarrhythmic agents should be continued.

3. Many of these patients are receiving digoxin and diuretics and will have electrolyte abnormalities. Any severe electrolyte abnormalities should be corrected.

4. Preoperative medication should be individualized for the patient's needs.

(b) Evaluating the pace-maker:

1. Non-programmable pace-maker:

The patient with a heart rate greater than the pace-maker rate should not have pace-maker impulses on the ECG. If pacemaker impulses occur just after the R-wave or in the T-wave, the pace-maker is malfunctioning. It is appropriate to call for the opinion of a cardiologist in this setting. Inserting a new generator could be part of the surgical procedure **(Kelly and Royster, 1989)**.

To evaluate a slowing intrinsic heart rate with a properly functioning generator, the patient should perform a Valsalva maneuver, the ring magnet should be placed over the generator changing the pace-maker to the fixed-rate VOO mode while observing for pace-maker impulses. Pace-maker spikes with capture should be apparent. The patient with an intrinsic heart rate less than the pace-maker rate should have clearly visible and regularly occurring pace-maker impulses associated with a peripheral pulse **(Kelly and Royster, 1989)**.

2. Programmable pace-maker:

- Interrogation of the pace-maker with appropriate programming device is the ideal. However, the examination described above usually sufficient .

- The program is determined by observing the preoperative ECG: Atrial and ventricular pacing impulses on the ECG then atrial and ventricular electrodes on the chest X-ray should be looked for **(Kelly and Royster, 1989)**.

Intraoperative management of the patient with a pace-maker:

(a) The pace-maker is not the problem. The real problem is the disease process that necessitated the pace-maker placement The patient should be monitored according to the underlying disease

1. Of critical importance is a monitor of pulsatility- either a finger on the pulse or a pulse oximeter. Perfusion can then be assessed even if the ECG is unreadable (e.g., during period of electro-cautery)

2. In Pulmonary artery catheters the fear of dislodging pacing wire is largely unwarranted as long as the pace-maker is ٤ to ٨ weeks old. The operator should be sensitive to alteration in resistance to catheter advancement. Additionally, the catheter should not be allowed to loop excessively in the ventricle.

(b) Induction and maintenance of anaesthesia can be performed with essentially all standard anaesthetic techniques. Pentothal remains a standard induction agent. A narcotic-relaxant technique and volatile agents can be used for maintenance.

Although not reported, propofol and ketamine can be used in patients with implanted pace-makers. Non depolarizing muscle relaxants and potent volatile agents should not interfere with pace-maker function. Regional anaesthetics are also acceptable.

(c) The automatic implantable cardioverter defibrillator (AICD) should be deactivated before use of the electrocautery but only after the patient is in the operating room and is fully monitored. It should be reactivated before leaving the operating room **(Levine, 1993).**

B. Direct current (DC) shock

Methods:

(a) Synchronized (cardioversion):

- The shock is triggered by the QRS complex on the E.C.G. This avoids delivering the shock during the vulnerable period of the T-wave.

(b) Asynchronized (defibrillation)

- The shock is delivered without regard to the E.C.G.

Indications

a. Ventricular fibrillation requires defibrillation.

b. Rapid ventricular tachycardia requires defibrillation when the individual QRS complexes are not easily distinguished from T-waves.

c. Atrial fibrillation, atrial flutter, SVT, and haemodynamically stable VT with distinct QRS are treated with cardioversion.

The degree of haemodynamic compromise usually determine the necessity and urgency for selecting cardioversion instead of drug therapy. Cardioversion is relatively contraindicated when digitalis toxicity is present and should be used with caution in patients with sinus node disease unless a pace-maker is in place. Serum K^+ should be normal when performed elective DC cardioversion **(Wellens and Brugada, 1995).**

III. General approach to treatment of arrhythmias

Assessment:

The effect of arrhythmias on hemodynamic performance is assessed primarily from measurments of arterial blood pressure and in some cases right and/ or left filling pressure. Treatment must be instituted promplty if the arrhythmias causes hemodynamic impairment **(Thomas and Krmer 1993)** .

Etiologic foctors:

Because even the most common intra operative arrhythmias are often atributable to simple, easily reversable causes , the usual etiologies should always be considered.

(1) Abnormal arterial blood analysis:

i) Hypoxia is a potent arrhythmogenic influence. It causes myocardial ischaemia directly through decreasing O_2 supply and indirectly stimulates catecholamine release causing increase in O_2 demand. Hypoxia initially causes tachycardia and finally bradycardia through direct depression on cardiac medullary centers.

ii) Hyprcarbia results in acidosis and increased sympathetic nerveous system activity causing tachycardia and ectopies.

iii) Hypocarbia results in respiratary alkolosis and K^+ shift (Serum K^+) intracelluarly.

iv) Electrolyte disturbances (particularly Ca^{++}, K^+): that result from massive fluid shifts, blood loss and acid base shifts.

2. Temperature:

1. hypothermia causes sinus bradycardia, atrial fibrillation and flutter.

2. Malignant hyperthermia is the most consistent cause of sinus tachycardia.

3. Ventricular arrhythmies are also frequently encountered **(Thomas and Krama 1995).**

3. Autonomic imbalance:

i) Sympathetic stimulation occurs with intubation, light anaesthesia, hypoglycaemia and hypovolemia.

ii) Parasympathetic stimulation, usually reflex in nature, is a common cause of bradyarhythmia.

iii) It may results from visceral traction. Laryngoscopy (in childern), carotid sinus massage, and extraocular muscle traction **(Thomas and Kramer 1993).**

4. Anaesthetic drugs:

i) Inhalational agents: Halothane sensitizes myocardium to catecholamines and causes ventricular arrhythmias and frequantly induce junctional rhythm.

ii) Muscle relaxants: Pancronium and Gallamine are vagolytic and, in addition, can stimulate adrenergic autonomic activity by blocking muscarinic inhibitory receptors located in sympathetic ganglia.

iii) Successive doses of succinycholine can result in sinus bradycardia, junctional rhythm, venticular arrhythmies and asystole**(Thomas and Kramer 1993)**

5. Myocardial factors either ischemia, congenital abnormal pathways, calcifications in conductive system, congenital or rheumatic valve disease.

Therapeutic Alternatives:

1. General approach:

It is reassuring to know that most arrhythmias are transient, cause no haemodynamic embarrassment, and resolve with the passage of time.

Maintenance of adequate oxygenation and ventilation, alterations of anaesthetic depth, maintenance of electrolyte balance, and relief of untoward reflexes are the usual modes of therapy. When circulatory compromise does occur, pressor support or even cardiac massage may be required, until normal sinus rhythm is restored **(Wellens and Brugada, 1995)**.

2- Slowing the heart rate:

a) Treatment of underlying cause (hypoxia , pain, full bladder, hypovolaemia, etc..).

b) Increase in anaesthetic depth (isoflurane may increase heart rate)

c) Vagal maneuvers (carotid sinus massage, valsalva).

d) Neostigmine 0.25 to 0.5 mg IV, may be used, although

the onset is slower and the duration is longer. It should be remembered that both these agents are anticholinesterases and may cause undesirable reversal of neuromuscular blockade.

e) B-blockade: For Sinus tachycardia propranolol, 0.5 to 1.0 mg IV, to a total dose of 0.1 mg/kg, or esmolol, 0.1 to 0.5 mg/kg be used. Similar doses are often required for re-entrant tachycardias involving the A-V node or slowing the ventricular response in atrial fibrillation.

f) Digoxin may be used for atrial fibrillation, flutter, and SVT. A knowledge of previous digitalis administration is essential.

g) Verapamil is especially useful in Slowing the ventricular response in atrial fibrillation or flutter and in terminating SVT. It can slow sinus rate and is the drug of choice in patients with reactive airway disease.

h) Adenosine may be used for terminating tachycardias in which the A-V node is part of he re-entrant circuit. It is also useful diagnostically for SVT of unknown type.

i) Overdrive atrial pacing is useful in treating some cases of SVT and atrial flutter.

j) DC shock is needed whenever rapid rate, ventricular

tachycardia or ventricular fibrillation causes severe haemodyanmic impairment **(Zipes, 1997).**

3- Increasing the heart rate:

a) Cessation of manipulations (ocular muscle traction, laryngoscopy, etc .).

b) Atropine, 0.4 to 2.0 mg IV.

c) Pancuronium and gallamine have a vagolytic effect.

d) Isoproterenol may be used as a drip, 0.5 to 2 mg/min. Increase in inotropy also occurs which when combined with chronotropic effect may significantly increase myocardial oxygen consumption; therefore, it should not be used in patient with ischaemic heart disease. Epinephrine or other catecholamine with B-adrenergic properties may be used.

e) Pacing **(Zipes, 1991).**

4- Treating ischaemia:

By optimizing heart rate, perfusion pressure, and ventricular volume.

5- Antiarhythmic drugs:

(As already mentioned previously)

(IV) Management of specific arrhythmias

Each arrhythmia is briefly described below, The emphasis is on pattern recognition with a classification system that should allow the anaesthesiologist to diagnose most of the arrhythmias seen in the operating room (Hurst et al., 1990).

A- Sinus bradycardia (shown in figure 6)

A. Diagnosis: Heart rate is < 60 bpm. P, QRS and T waves are normal.

B. Sinus bradycardia requires treatment if there is hypotension or escape rhythms, extreme bradycardia may allow a ventricular focus to take over and lead to ventricular tachycardia. The foot of the bed should be raised to assist venous return and atropine should be given IV, if persists the cautious use of isoproterenol may be employed **(Hurst et al., 1990).**

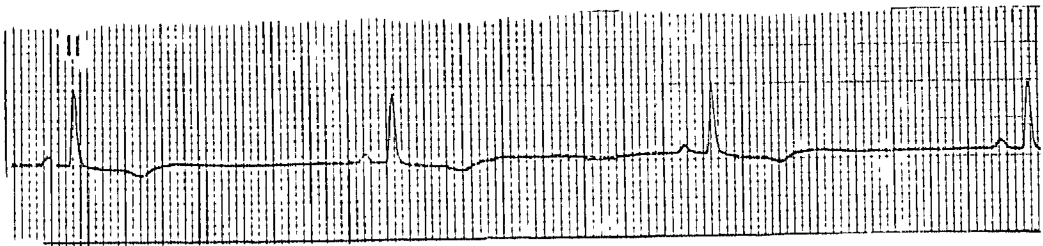


Fig. (6): Sinus bradycardia at a rate of 50 bpm **(From Hurst et al., 1990).**

B-Sinus tachycardia (shown in figure 7):

A. Diagnosis: Heart rate is > 100 bpm, P and QRS waves are normal. ST and T waves are normal but often with non specific changes.

B. Treatment: At first an attempt should be made to correct the underlying problem (lack of analgesia, lack of anaesthesia, hypoxia,...) then propranolol, verapamil, tensilon or neostigmine may be used. ECG and haemodynamic criteria should be used to decide if the rate is too fast for the patient's specific disorder (coronary artery disease, valvular stenosis, etc..) **(Hurst et al., 1990).**

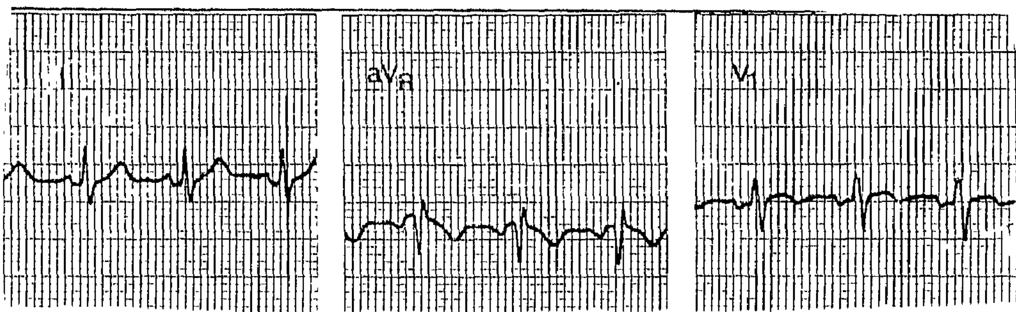


Fig 7: Tachycardia at a rate of 167 bpm Note the sinus P wave on the descending portion of the preceding T wave QRS is of normal duration and shows evidence of an old septal infarct (lead VI) **(From Hurst et al., 1990).**

C. Ectopic events:

1- Premature atrial contraction with normal conduction (shown in figures 8 and 9).

i) Diagnosis: The P wave is premature and abnormally shaped; the P-R interval is non diagnostic, the QRS and T waves are normal. There is usually a non compensatory pause. Occasionally the P wave is so early that it finds the A-V node refractory. If the B Wave is hidden in the preceding T wave and the QRS complex is absent, there is a pause that may mimic a sinus pause.

b) Treatment: Usually non, atropine or pacing for very slow underlying rate **(Zipes, 1988).**



Fig. 8: Atrial bigeminy. The arrows point to the premature atrial complexes. which are in a bigeminal pattern. Note that the premature P waves are different from those that appear in the normal beats and that the P-R interval is slightly prolonged **(From Zipes, 1988).**

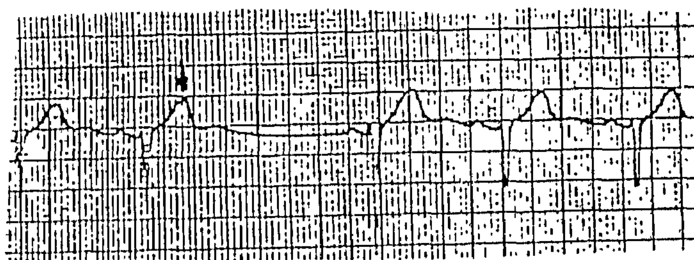


Fig. 9: Blocked piemature atrial contraction The arrow points to the premature P wave, which is not conducted and iesults in a slight pause before the next sinus beat The pause also results in a slight alteration in conduction, and the QRS complex is slightly different from the beats surrounding it **(From Zipes, 1988).**

2- Junctional arrhythmia:

a) Diagnosis: The P wave closely precedes, occurs within, or follows QRS and is often inverted in leads II, 111, aVF, QRS and T waves are normal. There may or may not be a compensatory pause

b) May be normal or due to anaesthetics or to autonomic imbalance.

c) Treatment: Usually non is necessary; alteration of anaesthetic depth may be helpful.

3- Premature ventricular contraction (PVC) (shown in figure 10):

a) Diagnosis: P-wave is absent (submerged in QRS) or occasionally retrograde after QRS. QRS complex is wide bizzare and premature. PVCs are most often followed by a full compensatory pause.

b) Treatment may include correction of underlying cause, deepening of anaesthesia, and/or use of lidocaine, procainamide or propranolol **(Zipes, 1988).**

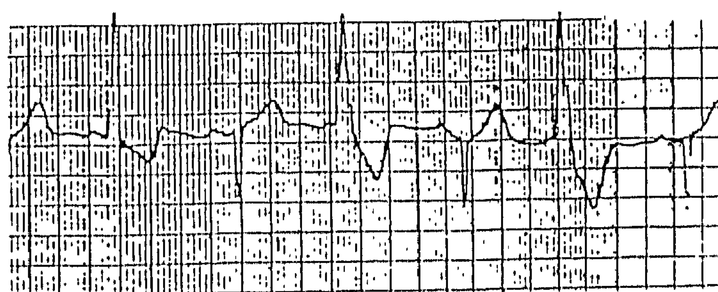


Fig. 10: PVCs in a bigeminal pattern. Note the wide, bizarre QRS with the ST segment sloping in the direction opposite to the main QRS deflection. A full compensatory pause is present **(From Zipes, 1988).**

D. Supraventricular arrhythmias:

1. Junctional rhythm (shows in figure 11):

a) Diagnosis: Heart rate is variable from 40 to 100 bpm. The P wave is absent or abnormal (usually inverted), closely preceding or following the QRS complex. QRS and T waves are normal

b) Treatment is alteration of anaesthetic depth When rate is low and associated with hypotension secondary to loss of atrial kick, atropine or ephedrine may be used in an effort to increase SA nodal activity. Vasopressor may be necessary to maintain blood pressure If heart rate is fast with maintained pressure, propranolol or verapamil may be used. **(Wellens and Brugada, 1995).**

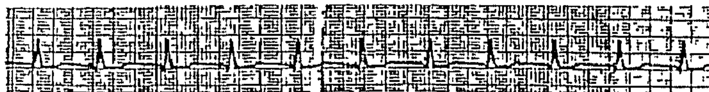


Fig. 11: Accelerated junctional rhythm-accelerated because the rate is 94 bpm, which is higher than the 60 to 65 the expected with the usual junction rhythms. No P waves are visible QRS and T waves are normal **(From Wellens and Brugada, 1995).**

2- Supraventricular tachycardia (SVT) (shown in figure 12):

a) Diagnosis: Heart rate is up to 250 P waves are abnormal; however, they are often merged with either

QRS or preceding T waves and not discernible. QRS complex is normal unless there is aberrant conduction, in which case a right bundle branch block (RBBB) pattern is usually present. ST and T wave changes may accompany this arrhythmia. It is often very difficult to distinguish SVT with aberrancy from ventricular tachycardia. Lead placement to optimize P wave amplitude may be helpful. If dissociation is noted, ventricular tachycardia is the very likely diagnosis.

b) Treatment: If vagal stimulation (by carotid massage) is unsuccessful, adenosine has the dual activity of being effective in most such tachycardies , while having no effect on a ventricular tachycardia. The response to adenosine is therefor of diagnostic value. If however the patient is in circulatory shock as a result of tachycardia, or drug treatment fails, a D C shock should be administered, for immediate effect. β-adrenoceptor blocker (e.g. sotalol, may be effective at preventing attack **(Wellens and Brugada, 1995).**

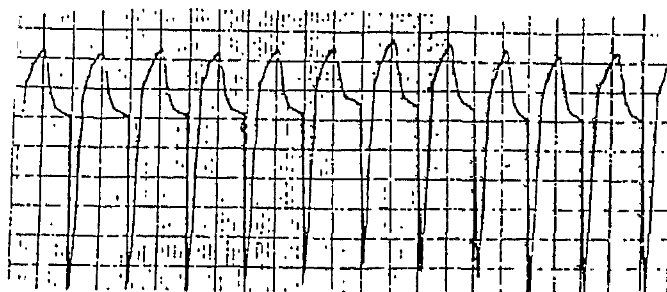


Fig. 12: Supraventricular tachycardia. The rate is 150 bpm. No definite P waves are discernible, and the QRS complex is narrow and lock regular. The exact site of origin, whether somewhere in the atrium or in the A-V junction, can't be determined from the scalar ECG; hence the generic term supraventricular tachycardia. **(From Wellens and Brugada, 1995).**

3- Atrial flutter: (shown in figure 13):

a) Diagnosis: Diagnostic features include an atrial rate of 250 to 350 bpm, a P wave with a saw tooth pattern, and not infrequently 2:1 AV conduction. Higher blocks (e.g., 4;1) and variable block also occur. The QRS complex is normal, although occasionally aberrant conduction is seen.

b) Treatment: Rapid atrial pacing may be attempted if atrial wires are in place. DC cardioversion is nearly 100 percent effective in converting this arrhythmia to normal sinus rhythm. Digoxin, propranolol, diltiazem, and verapamil, while occasionally terminating this arrhythmia, more commonly increase AV block and slow the ventricular rate. Type IA drugs are more likely to terminate this arrhythmia **(Wellens and Brugada, 1995).**

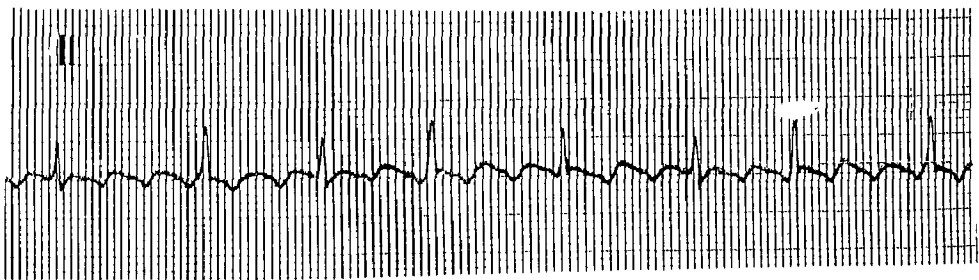


Fig **13:** Atrial flutter with variable A-V block Note the characteristic saw-toothed P-waves. The QRS complexes are narrow and are irregularly spaced. This is due to the variable A-V block than runs from 2:1 to 5:1 **(From Wellens and Brugada 1995).**

4- Atrial fibrillation (shown in figure 14):

a) Diagnosis; The ECG shows an undulating baseline, with a ventricular rate often 60 to 170 bpm. The complex is usually normal but occasionally irregular. Aberrant conduction may be seen when a short P-R interval follows a long interval. If the ventricular rate become regular, digitalis toxicity should be considered.

c) Treatment: Digoxin, propranolol diltiazem, and verapamil may be used for control of ventricular response. Procainamide may terminate this arrhythmia If the patient decompensates with loss of atrial kick and the onset of atrial fibrillation is acute, cardioversion is the treatment of choice **(Wellens and Brugada, 1995).**

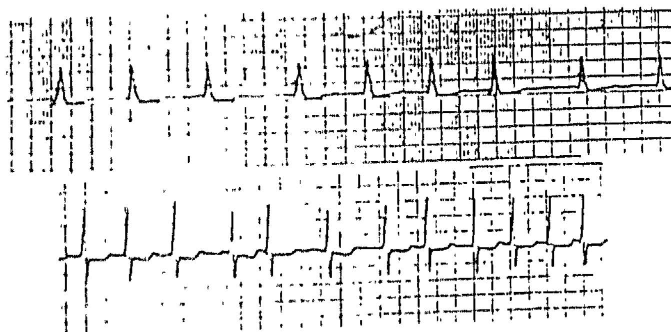


Fig. 14: Atrial fibrillation Note the slight undulation of the baseline without discernible P waves The QRS complexes ace normal and are irregularly spaced. The top tracing shows a slow ventricular response of 75 bpm, the bottom tracing shows a response of 120 bpm **(From Wellens and Brugada, 1995)**

5- Wandering atrial pacemaker;

a) Diagnosis: Heart rate < 100 bpm P-wave of variable morphology, normal QRS complex.

b) Treatment: Non is indicated, as this condition is benign **(Wellens and Brugada, 1995).**

6- Multifocal atrial tachycardia:

a) Diagnosis: Heart rate is >100 bpm. the P wave has at least three different waveform, with an irregular P-P and a variable P-R interval; and the QRS complex is normal.

b) Treatment is aimed at improving pulmonary status (**Kastor, 1990).**

E. Ventricular arrhythmias:

1. Premature ventricular contractions (as already mentioned previously).

2. Ventricular tachycardia (VT), (shown in figure 15);

a) Diagnosis: This Arrhythmia occurs when three or more PVCs occur in succession with a rate greater than 100 bpm. The P wave may not be seen but will sometimes march through or be conducted retrograde. The QRS complex is wide and bizarre.

Treatment:

- Sustained VT should be treated.
- Non sustained VT is treated only when it is associated with active cardiac disease.
- If the patient is unstable use DC counter shocks, starting at 50 joules increase by 50 joules each time as needed.
- If the patient is stable, start lidocaine as a bolus and a continuous infusion, if this is ineffective and the Q interval is normal, consider intravenous amisdarone. This has proven effective for refractory arrhythmias.

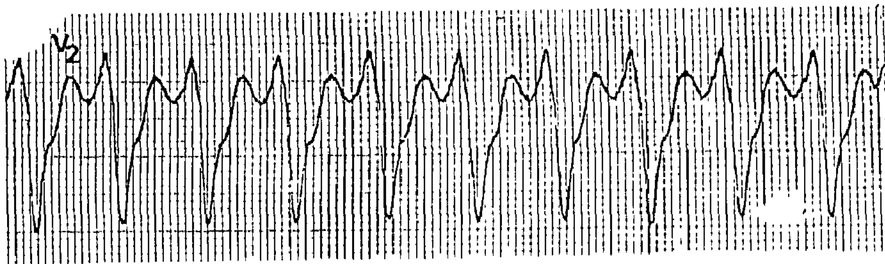


Fig. 15: Ventricular tachycardia the QRS complexes are wide and bizarre **(From Hurst et al., 1990).**

3- Ventricular fibrillation (shown in Figure 16):

a) Diagnosis: The ventricles discharge in a completely chaotic fashion and no QRS complex is seen on the ECG.

b) Treatment is DC shock; to maintain rhythm after the shock, lidocaine, procainamide, and/or bretylium are used.

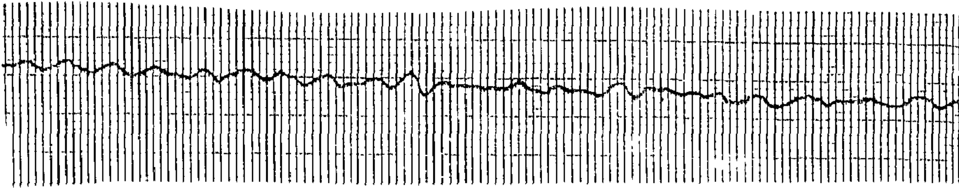


Fig. 16 : Ventricular fibrillation **(Hurst et al., 1990).**

F- Wide ORS Complex:

1. Aberrant conduction (shown in figures 17):

a) Diagnosis: The P-wave may be normal (sinus tachycardia) or abnormal (premature atrial contraction atrial fibrillation and flutter). The QRS usually shows the RBBB pattern. No compensatory-pause is seen.

b) Treatment: Non is needed unless the underlying rhythm is atrial fibrillation or flutter in which case, the rate should be controlled **(Marriott, 1996).**

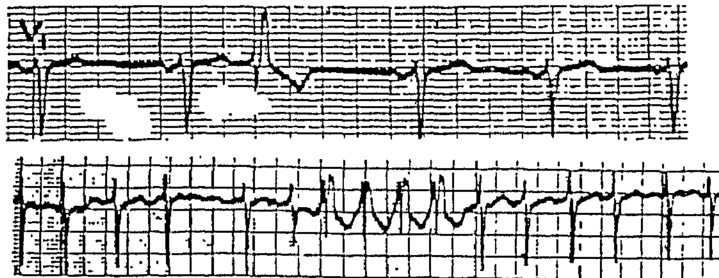


Fig. 17: Premature atrial contraction with aberrant conduction. The abnormal P wave is not visible. The QRS complex shows an RSR' prime pattern in cad V_1, Note the short interval from, lie sinus beat to the ectopic beat compared with the normal R-R interval **(From Marriott, 1996).**

2- Premature ventricular contraction (as already mentioned previously).

3- Bundle branch block (shown in figure 19):

a) Left bundle branch block (LBBB):

1. Diagnosis: The P-wave is normal The QRS complex is prolonged to ≥ 0.12 seconds; and a wide nonphasic or notched R wave is typically present in leads. I, V_5, and V_6 with QS or a small R with large S in V_1 .

2. Treatment None **(Marriott, 1996).**

b) Right bundle branch block (RBBB).

1. Diagnosis; The P wave is normal. QRS is prolonged to ≥0.12 seconds. Typically RSR' is present n lead V_1 and a wide S in leads I, 'V5 and V_6. The condition can occur during passage of pulmonary artery catheter.

2. Treatment: There is no treatment, but one must rule out pulmonary embolus if acute RBBB occurs intraoperatively **(Marriott, 1996).**

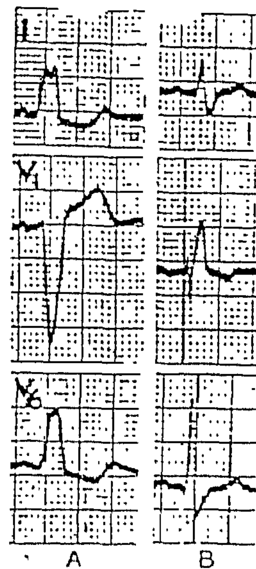


Table 19: (A) left and (B) right bundle branch block. This figure shows the important leads to look at in the diagnosis of bundle branch blocks I, V_1, and V_6 **(From Marriott, 1996).**

4- Wolff-Parkinson-White (WPW) syndrome:

a) Diagnosis: Normal P wave, shortened PR interval with prolonged QRS secondary to slurred upstroke.

b) Treatment Narrow complex tachycardia may respond to adenosine, propanol, or procainamide. When the arrhythmia is associated with hypotension, DC cardioversion may be necessary Verapamil and digoxin may convert the tachycardia to AF with rapid response and should be avoided.

5- Ventricular tachycardia;

(As already mentioned previously).

F. Atrioventricular block:

1. First degree A-V block (shown in figure 20):

a) Diagnosis; Normal P wave; P-R interval>0.20 seconds, normal QRS complex.

b) Treatment: None is indicated **(Thomas and Kramer, 1993).**

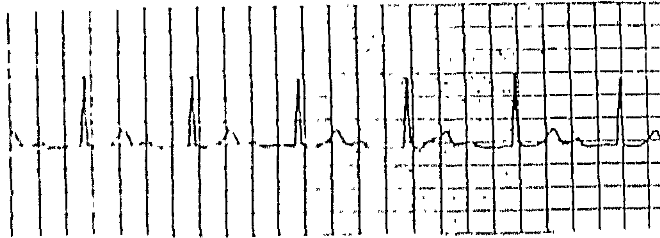


Fig. 20: First degree block. Note the prolonged PR interval of approximately 400 ms. (From Thomas and Kramer, 1993).

2- Second degree A-V block:

a. Mobitz I (Wenckebach); (shown in figure 21):

1) Diagnosis: The P wave is normal. There is a progressive increase in P-R interval the P wave is not conducted. This is followed either by resumption. of cycle or by a junctional or ventricular escape beat. The QRS complex is normal unless there is a

coexisting bundle branch block.

2) Treatment is usually unnecessary. For extremely slow rates, pacing maybe indicated (Thomas. and Kramer, 1993).

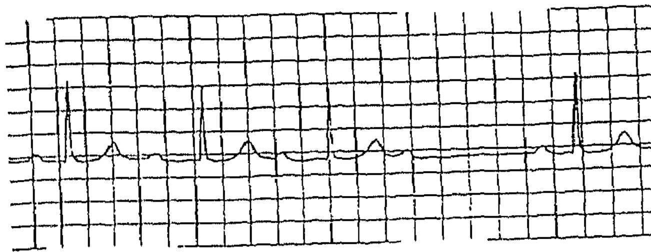


Fig. 21: Second-degree Mobitz type I heart block. The PR interval becomes progressively longer until one QRS is dropped ((**From Thomas and Kramer, 1993**).

b. Mobitz II (shown in figure 22) :

1) Diagnosis: Consecutively conducted beats occur with normal P waves and constant P-R intervals before the dropped beat With advanced block, multiple P waves are present.

2) Treatment : Ventricular pace-maker **(From Thomas and Kramer, 1993).**

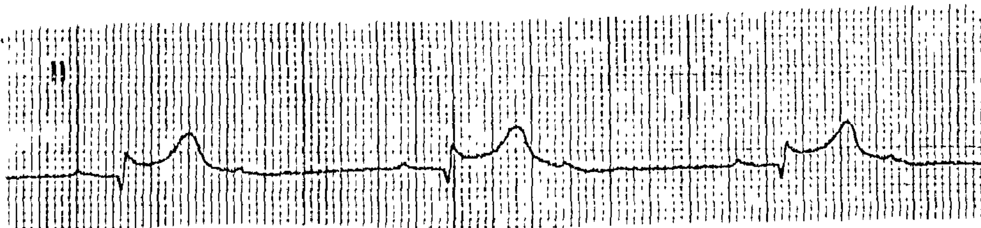


Fig. 22: Second-degree Mobitz type II heart block. In the example every other QRS is dropped **(From Thomas and Kramer, 1993).**

3. Complete heart block (shown in figure 23)

a) Diagnosis: Heart rate is 30 to 40 bpm. The P wave shows normal morphology but no relation to QRS complex. The QRS complex is wide, regular, and totally independent of the P wave

ii) Treatment: Atropine or isoproterenol to increase ventricular rate until pacing can be initiated (Thomas and Kramer, 1993).

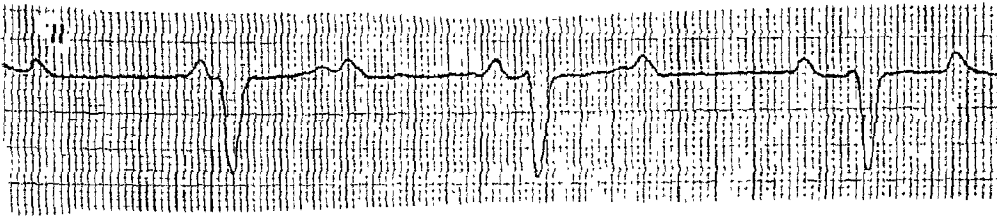


Fig. 23: Third-degree (complete) heart block. The P-waves are not conducted into the ventricle (**From Thomas and Kramer, 1993**).

Summary

SUMMARY

Cardiac dysrhythmia is one of the most prevalent complications that face the anesthesiologists in the perioperative period.

However, the incidence appears higher with the computer-aided detection of dysrhythmias, coexisting cardiovascular, cerebrovascular, ulmonary, enal, metabolic or electrolyte disturbances. Also it shows a further increase in patients receiving concurrent drug therapy as digitalis, diuretics and drugs affecting the synthesis and release of catecholamine

The sudden appearance of any new dysrhythmias, regardless the haemodynamic consequences should arouse the anaesthesiologist interest and concern. However, more specifically important are dysrhythmias of haemodynamic significance which are likely to lead to more life threatening rhythm disturbances. Among those advanced second or third degree heart block which may lead to bradycardia, asystole or unstable escape rhythm. R on T frequent ventricular ectopics that lead to ventricular

tachycardia or fibrillation, any persistent supraventricular or multiform ventricular ectopics.

The successful dysrhythmia management requires some familiarity with cellular mechanisms for cardiac dysrhythmias since the most specific management is antidysrhythmic drugs or electrical therapy needs a deep knowledge about these mechanisms including cardiac action potential, the normal and abnormal automaticity.

The possible management of dysrhythmias should be directed towards the cause including possible hypoxia, hypercarbia, electrolyte disturbance, myocardial ischaemia or infarction, anaesthetic overdose, hypertensive or hypotensive episodes or possible drug interaction

The anaesthetic management for dysrhythmias should start in the preoperative period by a thorough preoperative assessment of the case for possible cause of dysrhythmias its or impact on haemodynamic status, drug therapy or the indication for pacing.

The intraoperative monitoring aids include the

routine 5 lead EGG with ST segment analysis, capnography, oxygen saturation, C.V.P. and P.A.W..P.

Among the new monitoring aids includes continuous blood gas analysis, trans-oesophageal echocardiography and transcutaneous echo-probes.

The postoperative follow up for possible reactivation of re-entry circuit should also be considered

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Arabic Summary

الدم بالأكسجين، جهاز قياس نسبة ثانى أكسيد الكربون. وتظهر أهمية قياس ضغط الدم فى الأوردة المركزية وضغط الدم بالشريان الرئوى فى حـــالات الاختلالات الناتجة من أمراض بصمامات القلب.

وقد أدخل حديثاً أجهزة قياس غازات الدم المستمر والموجات فـــوق الصوتية على القلب من خلال المرئ أو من خلال الجلد.

إن خطة طبيب التخدير أيضاً لابد وان تشتمل على وعى بالأدويـــة التى يستعملها المريض وإمكانية حدوث تداخل بينها وبين أدويـــة التخديــر والتى قد تؤثر سلبياً على توصيل الشحنات بالقلب أو على نشـــأة الشـــحنة الكهربائية ولابد أن يوضع فى الاعتبار المتابعة فى فترة ما بعد الجراحـــة، حيث أن هذه الفترة قد تشهد نشاط للبؤرة المؤدية إلى اختـــلالات القلـــب أو نشوء الدائرة الكهربائية المؤدية إلى اختلالات خطيرة.

زيادة النبضات البطينية أو الرعشة البطينية. من بين الاختلالات الخطيرة أيضاً اختلال الضربات الأذينية المستمر أو الاختلال البطينى متعدد الأشكال.

إن العلاج الناجح للاختلالات فى ضربات القلب يتطلب نوعاً من التألف مع النظم الخلوية لحدوث هذه الاختلالات حيث أن العلاج الأساسى الذى يختص بهذا الأمر هى الأدوية المثبطة لاختلالات القلب أو العلاج الكهربائى وكلاهما يحتاج إلى معرفة عميقة بفسيولوجية نقل الشحنة الكهربائية بالقلب بالصورة الطبيعية وكيفية حدوث الاختلال.

أن التصرف فى الفترة ما حول إجراء الجراحة لابد وأن يتوجه ناحية تحديد السبب والذى قد يكون نقص نسبة الأكسجين بالدم، زيادة نسبة ثانى أكسيد الكربون بالدم، اختلال إلكتروليتات الجسم، قصور الشريان التاجى، زيادة جرعة التخدير أو نوبات زيادة أو نقص حاد فى ضغط الدم.

إن الخطة التى يعدها طبيب التخدير تشمل البحث الدقيق للحالة لمعرفة السبب المحتمل لهذه الاختلالات وأثرها على الدورة الدموية الحركية، وأيضاً جميع الأدوية التى يتعاطاها المريض كما لابد أن يبحث طبيب التخدير إذا كانت هناك أى مداعاة لوضع منظم لضربات القلب قبل الجراحة.

أما عن أجهزة متابعة المريض تحت التخدير فتشمل جهاز متابعة ضربات القلب المجهز بخمسة أطراف وجهاز التقاط الاختلالات وإمكانية وجود محلل لمنطقة الـ ST فى رسم القلب، وأيضاً جهاز قياس درجة تشبع

الملخص العربى

يعد اختلال ضربات القلب واحد من أكثر المضاعفات شيوعاً والتى تواجه أطباء التخدير فى فترة ما حول إجراء التدخلات الجراحية.

وفى دراسة تم عمل حصر لهذا النوع من المضاعفات فكانت تمثل حوالى من إجمالى الحالات. بيد أن هذه النسبة بدت أكبر منذ استخدام الكمبيوتر فى ملاحظة وملاحقة حدوث اختلال ضربات القلب وفى الحالات التى تعانى مسبقاً من الأمراض التالية: أمراض القلب والأوعية الدموية أمراض المخ وأوعيته الدموية، أمراض الجهاز التنفسى، أمراض الكليتين، اختلال الأيض وإلكتروليتات الجسم. كما تزيد النسبة فى حالات المرضى الذين يتعاطون عقاقير مثل الديجيتاليس، مدرات البول، والعقاقير المؤثرة على تصنيع أو إخراج مادة الكاتيكولامين.

الظهور المفاجئ لأى اختلالات فى ضربات القلب، بغض النظر عن كونها مؤثرة أو غير مؤثرة على الدورة الدموية الحركية لابد وأن تسترعى انتباه طبيب التخدير ولكن الأخص بالاهتمام هى اختلالات القلب المؤثرة على الدورة الدموية الحركية والتى قد تؤدى إلى اختلالات قد تهدد حياة المرضى.

بين هذه الاختلالات الأنواع الآتية: الدرجة الثانية المتقدمة أو الدرجة الثالثة من تثبيط نقل كهربية القلب والتى تؤدى إلى بطء فى ضربات القلب أو توقفه تماماً أو عدم الإحساس بالنبض لفترة وجيزة. أيضاً وجود الـ R على T فى رسم القلب، تعدد اختلال الضربات البيطنية والتى تؤدى إلى

اضطرابات نظم القلب أثناء العمليات الجراحية
الأسباب والمعالجة

رسالة

توطئة للحصول على درجة الماجستير

في التخدير

مقدمه من

الطبيب/ أسامه محمد محمد غنيمى

تحت إشراف

أ.د. يحيى عبد الرحيم هميمى

أستاذ التخدير والرعاية المركزة

كلية الطب- جامعة عين شمس

أ.م. مجدى محمد حسين نافع

أستاذ مساعد التخدير والرعاية المركزة

كلية الطب- جامعة عين شمس

د. هشام محمد محمود طاحون

مدرس التخدير والرعاية المركزة

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